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LABORATORY OUTLINES

IN

PLANT PATHOLOGY

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COMPLETELY REVISED AND REWRITTEN
BY THE SENIOR AUTHOR

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PREFACE

THESE outlines are intended for students taking a regular four-year course in college or university. They are designed for the purpose of effectively acquainting the student with the laboratory materials presented and are the result of years of experience and test in actual practice work. As here presented these outlines constitute a complete revision of those published in the original edition of 1916. Some exercises included in that edition have been omitted and some new ones substituted. While some radical changes in arrangement and terminology have been introduced, the general character of the outlines remains the same. For all these changes full responsibility lies with the senior author.

Although the acquisition of a body of facts is an important and necessary part of the work in such a course, a more vital feature is the training in logical methods of acquiring them. The student is urged to follow his outline carefully, making sure at each step that the outline and the materials before him agree. The same sequence of treatment is followed throughout all the exercises on the different diseases. This sequence in procedure should be mastered promptly. The term paper will afford an opportunity for determining how well the student has grasped the logic of this procedure.

The grouping of diseases here presented is, I believe, an important step in calling attention to the domination of systematic mycology in phytopathologic teaching and writing, and of directing it toward the more logical classification and study of diseases on the basis of the pathologic phenomena exhibited. At the same time the subgrouping of the diseases provides for an introduction to the chief groups of etiologic agents.

It is not expected that all the diseases herein outlined will be covered in the average college course. Experience has shown that fifteen to twenty of these exercises are all that can be effectively mastered in a one semester three-hour course. The instructor will make such selections from the different groups as will best serve his purpose in illustrating the fundamentals of the subject in the case of the students in his classes. No laboratory practice in the methods of control of the diseases studied is provided. That phase of the subject is fully treated in the course based upon and following this, namely, The Principles of Plant Disease Control.

These outlines are designed specifically for the work as given at Cornell University, but it is hoped that teachers elsewhere may find them usable in their classes. The text supplementary to the laboratory work, now in mimeograph form, is being prepared for publication.

Acknowledgment is due to all those who through the years have assisted with suggestions and criticisms in the formulation and perfection of these

outlines. I am especially indebted to the three whose names appear on the title page as co-authors. Their personal assistance in the preparation of the first edition has made this revision relatively simple and easy. To Professor Hesler I am under special obligations for criticisms and suggestions in the preparation of the manuscript of the present revision.

H. H. Whetzel.

Cornell University, Ithaca, N. Y.,
January, 1925.

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LABORATORY OUTLINES
IN
PLANT PATHOLOGY

SYMPTOMATOLOGY

This exercise is designed to acquaint the student with the names and characteristics of the more common symptoms and signs exhibited by diseased plants.

Since the identification of diseases, especially in the field, must depend largely upon familiarity with the signs and symptoms characteristic of each disease, a knowledge of the types of these is fundamental to progress in the study of plant pathology. Symptoms may best be studied in plants in the fresh or growing condition since color and attitude of affected parts are very important features in many types of symptoms and both are often altered in preserved materials. The study of signs and symptoms in the field is the ideal method, but the limitations set by the time available in a college course largely discount the field-trip method under the conditions. Several trips covering extended areas during one or more seasons would be required to provide materials which can be made available by the plan to be followed here.* The student should supplement this exercise by several field excursions taken on his own initiative. Upon the request of five or more students short trips to fields, woods and gardens in the vicinity may be conducted by the professor or one of the instructors giving the course. Such trips should be arranged before frost has destroyed the vegetation.

LABORATORY STUDIES

1. Study carefully the classification and definitions of the symptoms given in the text on this subject.
2. Examine carefully each of the numbered specimens displayed on the laboratory tables. Determine the most outstanding symptom exhibited by each specimen.
3. List the displayed specimens by number, indicating the data called for in the following tabular form.

Symptoms				Signs		
No.	Group	Subclass	Name	Group	Class	Name

---

* A numbered set of specimens (75–100) showing as many types of symptoms and signs as possible are arranged in the laboratory for inspection and study. Fresh material (so far as available), preserved specimens and photographs are used.

4. Sketch carefully, from the specimens displayed, to illustrate ten different types of symptoms and five different signs. It will be profitable to color the sketches with crayons or water-colors to show color characters. Name and classify each, giving:—group, class and subclass to which it belongs. Write a brief description of each.

5. When studies have been completed on this exercise, report for conference by writing your name on the card provided for the purpose in the laboratory. In your turn you will be called by one of the instructors for this conference in the adjoining laboratory. Take with you all notes and drawings.

6. The conferences will be in the nature of a test of your ability to recognize and classify signs and symptoms of plant diseases as a result of the studies made. For this test, four sets of specimens and photographs will be displayed.

Group *a*. Fresh and preserved specimens in jars. Sort these into the three symptom groups on the basis of the most prominent symptom exhibited. This should be accomplished in less than ten minutes.

Group *b*. A numbered set of specimens showing different types of *symptoms*. Make a list of these giving:—

No.	Name of symptom	Group	Class	Suscept organ

Group *c*. A numbered set of specimens showing different types of *signs*. Make a list showing:—

No.	Name of sign	Group	Class	

Group *d*. Colored photographs of diseased plants. The instructor will give you an oral test on these.

In addition to these, two other items will be considered in making up the grade on this exercise: the identification list prepared in accordance with (3); and drawings and descriptions prepared as indicated in (4).

THE LITERATURE OF PLANT DISEASES

The literature on plant diseases is widely scattered. Abstracts of the current literature on the subject (American and foreign) appear regularly in each number of the EXPERIMENT STATION RECORD and BOTANICAL ABSTRACTS. Other journals and publications in which such reviews and abstracts of phytopathological papers are to be found, follow:—

Review of Applied Mycology.
Zeitschrift für Pflanzenkrankheiten.
Botanishes Centralblatt.
Centralblatt für Bakterilogie und Parasitenkunde.
Just's Botanische Jahresbericht.
Index au Bulletin Bibliographique Hebdomadaire Institut International d'Agriculture.

Texts and reference books in which references to the literature of the subject are more or less brought together, are:—

Butler, E. J. Fungi and disease in plants. 1918.
Duggar, B. M. Fungous diseases of plants. 1909.
Hesler, L. R., and Whetzel, H. H. Manual of fruit diseases. 1917.
Stevens, F. L. The fungi which cause plant disease. 1913.
Rankin, W. H. Manual of tree diseases. 1918.
Smith, E. F. Bacteria in relation to plant diseases. Vol. I–III. 1905, 1911, 1914.
Smith, E. F. Bacterial diseases of plants. 1920.
Massee, G. Diseases of cultivated plants and trees. 1910.

PROCEDURE IN PREPARING A BIBLIOGRAPHY

Choose a disease from the list of term paper subjects, in the term paper outline, according to directions there given.

Key-words sheet. Write in three different columns on one of the 5 x 8 reference sheets provided: (*a*) the names of the suscepts; (*b*) different names applied to the disease; (*c*) different names applied to the pathogene.

Note:—Begin each column with the names given in the subject list, adding to each, the other suscepts, other names of the disease and other names of the pathogene, discovered as the work proceeds. These will constitute *key words* under which references to the disease in hand will be looked for in the indices.

Check list. Select the *latest* volume of the Experiment Station Record available, and, turning to the index, locate under key words the references to the disease chosen.

1. Write on a sheet of paper (the check list) the number of the volume in hand.

2. Record under this volume number, in numerical order, the pages on which references to the disease are to be found.

Suppose for example the key word as found in the subject list for the term paper are:—

Apple *Bitter Rot* *Glomerella cingulata*

First consult the index under "apple," noting the page of every item that may possibly refer to the disease, bitter rot, and record as directed above.

Then consult the index under "bitter rot," for one may find there references to the disease not listed under "apple."

Finally, consult the index under the names of the pathogene.

3. Now examine each reference in the text carefully, copy according to directions below, any of the references which refer in any way to the disease.

Directions for copying references. Copy the references on the 5 x 8 sheets of paper provided, using *one sheet for one reference only. Begin at the very top.* Write *lengthwise* of the sheet and arrange the data as follows:—

4. Record the source of the reference in the *very upper right-hand* corner thus:—E. S. R. **18** : 748. (See Information on "Source of reference," below.)

5. Place the surname of the author, followed by his initials in the *upper left-hand* corner on the line below that of the source of the reference.

6. Directly following the author's name record in order:—(title) (period) (publication) (series) (colon) (vol.) (part or number) (colon) (pages) (comma) (plates) (comma) (figures) (period) (date of publication) (period). See sample sheet below.

Note:—No matter what the arrangement and punctuation given in the source of the reference or in the original, it should be rearranged and punctuated as above.

When in doubt on any point, consult "Information" (below) under the proper heading.

SAMPLE-SHEET

E. S. R. **47** : 275

Hasselbring, H. Bitter rot. Agri. Sci. Jour. 2 : **47^3** : 37–47, pl. 1–4, fig. 1–9. 1906.
(Abstract from original)

SUSCEPTS

Suscepts listed are apple, pear, etc. p. 37.

Variety Susceptibility

Most susceptible, Greening, Baldwin; least susceptible, Winesap, etc. p. 39.

DISEASE

Name

Following names listed; bitter rot, anthracnose, ripe rot, p. 40. (Continue on other sheets under the pertinent heads and subheads as used in the outline for the term paper.)

INFORMATION

Source of reference. Having the source with each reference will enable one readily to verify it or correct errors made in copying. It should include the abbreviated name of the publication, volume-numeral (or of year or number as the case may be), colon (:), followed by the page on which the reference occurs. If the article is first discovered in the original, indicate thus:—"Orig.".

Author. Surname, comma, christian name or initials (just as cited in the original), period, is the order of the arrangement. Surnames with "von," "de," and the like write thus:—"Liebenberg, A. von." Where there are two or more authors, arrange each name in the same way connecting the names with "and" or its foreign equivalent, or with commas and "and" in the case of more than two authors. In the case of several authors one may write for example "Stewart, F. C. and others" or "Stewart, F. C. et al." In the case of anonymous articles write in place of the author's name the word (Anonymous), enclosed in parentheses.

Title. The title of an article follows directly after the author's name and should always be in the language of the original if possible. When only the translation of the title is given precede it with the name of the original language and the word "title" in brackets, thus:—"(Italian title). Concerning the influence, etc.". When both the original and the translation of the title are given, the translation follows the original and is to be included in brackets. No abbreviations in the title should be used except such as appear in the original. Only the first word and proper names in the title may be capitalized, except when the title is in German in which case all nouns are capitalized. The title terminates with a period.

Name of society or organization publishing the work follows directly after the title, using a second line, if needed, beginning directly under the fourth letter of the author's surname. It is followed by a period; abbreviations allowed.

Name of publication. Journal, Berichte, bulletin (properly abbreviated) follows (preceded by a space), directly after the name of the society or organization, or where these are wanting, directly after the title. When the last word is not an abbreviation no punctuation mark follows.

Periodicals. The name of periodicals are usually abbreviated, but only the first word is capitalized and is not separated by punctuation from the volume numeral thus:—"Gard. and for. **8** : 28" or "Hedwigia **9** : 23."

Bulletins, circulars, memoirs, leaflets and the like are usually numbered and are not considered as constituting consecutively paged numbers of a volume. The numerals appear therefore in place of the volume numerals, but *are not to be underscored*. The proper abbreviation for the word, Bulletin, is Bul. with a capital B. Where qualified, as for example, Technical bulletin, it is abbreviated but not capitalized, thus:—"Tech. bul."

Books. In cases where no volume is given, cite the pages thus:—"p. 27 or p. 1–441."

Citation from books. Following the author's name give the title of the chapter or paragraph, especially referred to, period (.), then *In*, then the title of the book followed by the letter p. to indicate pages, then the page numbers and finally the date, Thus:—"Duggar, B. M. Brown rot of stone fruits. *In* Fungous diseases of plants, p. 187–194. 1909.

Government publications are always to be cited in the following form:—U. S. Agr. Dept. Yearbook; or U. S. Plant Ind. Bur. Bul. 37 : 23–48. 1909.

State Experiment Station Publications are always to be cited thus:—Alabama Agr. Exp. Sta. Bul.; or New York (Geneva) Agr. Exp. Sta. Rept.; Cornell Univ. Agr. Exp. Sta. Bul.

Volume numerals are always to be in arabic and are to be **underscored with a wavy line, indicating bold-faced type in printing.** The word volume **or its abbrevaition** (vol.) *should not* appear in a reference.

Reports are often issued for a given year as for example the Alabama Agr. Exp. Sta. Report for 1896, no volume number being given. In such a case the *year numerals take the place of volume numerals* and the above would appear thus:—Alabama Agr. Exp. Sta. Rept. **1896** : 1–87.

Number numerals when indicating consecutively pages parts of a volume (as for example, vol. 27, No. 6, pp. 70–83) *should not* appear in the reference. When not paged consecutively the number numeral appears as a superior figure to the right above the volume numeral thus:"$\mathbf{27}^6$: 70–83."

Series, Abteilungen and the like. If a periodical is issued in two or more separate series the *series numeral precedes the volume numeral*, separated from it by a colon and is not to be underscored as:—"3 : **46** : 23–87." Sometimes the series is indicated thus:—"Science, n. s. **42** : 47–56." The n. s. = New Series.

Parts. If parts of a volume are paged separately, the part numeral *follows* the volume numeral in a superior position thus:—"$\mathbf{82}^2$: 241–256" (same as number numeral which see).

Pages. Where the page numerals are preceded by volume numerals or number numerals and a colon, the pages are to be cited inclusively, i. e., first and last pages separated by a dash, thus:—"47–83", and followed by a period unless followed by plates or figures in which case a comma follows the page numerals.

Tables, plates, and **figures** (properly abbreviated, tab. pl. fig.) follow directly after the pages, followed by the proper numerals in arabic and comma or period, depending upon their position with respect to the date. They are, like pages, to be cited inclusively.

Year. This must be the actual year of publication. It may not always be determined with certainty except by consulting the original. Exercise care on this point and leave the space blank until the original can be consulted, if not absolutely sure of it from the reference.

The method of citing references outlined above is that used in the College of Agriculture, Cornell University. For further details see "Notes for the guidance of authors," an unnumbered pamphlet issued by the editorial office of that college.

For other methods of citing literature see references below.

PREPARATION OF AN ABSTRACT OF AN ARTICLE

The abstract should always be made from the original, unless for some good reason this is impossible. Do not copy the abstract of the article as it appears in the Experiment Station Record or elsewhere.

Begin the abstract on the reference sheet, two or three lines beneath the reference. Use both sides of the paper or use additional sheets or both if desired.

One should abstract as he reads, assorting and entering data under the proper head as indicated in the outline for writing term paper. (See term paper outline and the sample sheet in this outline.)

Follow each entry of data with the number of the page on which it was found in the original. This will enable one when writing the term paper to readily cite the source of any statement made.

The abstract should be full enough so that it will not usually be necessary to refer again to the original in preparing the term paper.

For conference or report. Hand in the bibliography of the subject chosen, arranged as follows:—

1. The references, each on a separate sheet. Arrange the references in the order of their importance.
2. One or more of the most important references carefully abstracted as described above.
3. In a folder labeled thus:—

(Name of suscept) (Name of disease) Bibl.

(Name of student)

References. Notes for the guidance of authors. New York State College of Agriculture at Cornell Univ. (Unnumbered pamphlet).

A guide for collaborators in the preparation of abstracts for Botanical Abstracts. Unnumbered pamphlet compiled by the Bibliographical Committee of Botanical Abstracts. Ithaca, N. Y., 1919 (see p. 3–4).

TERM PAPER

The term paper exercise consists in preparing a short paper on one of the diseases listed. The paper should be essentially a text on the disease chosen.

INFORMATION

1. The instructor will designate those subjects in the appended list from which selections for each term paper are to be made. Any disease in the designated list may be chosen. The same subject may be chosen by more than one student, if approved by the instructor, but it is generally to the student's advantage for as few as possible to be working on the same subject because of the limited number of available copies of some of the articles to be consulted.
2. Students choosing the same subject will be expected to work independently in consulting the literature and preparing the paper.
3. If so desired, the paper may be illustrated. This will add to its value. Illustrations may be in the form of text figures or plates, either drawings or photographs.
4. The student may write on some disease not listed herein, if permission is granted by the instructor.
5. The selection of a subject for the term paper will be made at the beginning of the laboratory exercise on The Literature of Plant Diseases.
6. The work in the laboratory will consist in obtaining references (provided for in the exercise on Literature of Plant Diseases) and getting such information and assistance from the instructor as may be needed to make clear the method of procedure. The reading, abstracting and writing of the paper is to be done outside regular laboratory hours.

PROCEDURE

After having located and listed the references bearing on the subject chosen, as outlined in the exercise on The Literature of Plant Diseases:—

7. Select the more important articles (usually the most recent).
8. Read and abstract these carefully according to directions given in the outline on The Literature of Plant Diseases.
9. If specimens and materials of the disease chosen are available, they may be studied in addition to the literature consulted.
10. Spread out the abstract and laboratory notes and proceed to correlate and arrange the data in each, according to the following outline:—

OUTLINE FOR ARRANGING DATA IN TERM PAPER

(TITLE)

SUSCEPTS

PLANTS AFFECTED

VARIETAL SUSCEPTIBILITY

DISEASE

NAMES

HISTORY AND RANGE

IMPORTANCE

SYMPTOMATOLOGY

Morphologic Symptoms ....
Signs ....
Histologic Symptoms ....

ETIOLOGY

Name, history and classification of the pathogene ....
Pathogenicity ....
Life history ....
  The Primary Cycle ....
    Pathogenesis ....
      Inoculation ....
      Incubation ....
      Infection ....
    Saprogenesis ....
  The Secondary Cycles ....(If secondary cycles requiring special consideration occur, repeat headings as for primary cycle.)

EPIPHYTOLOGY

CONTROL

EXCLUSION

ERADICATION

PROTECTION

IMMUNIZATION

BIBLIOGRAPHY

11. The heads and subheads indicated in the above outline are quite definitely fixed and will be the same for any disease caused by a pathogenic organism. They should appear, properly placed, in the report submitted.

12. For diseases not due to pathogenes the subheads under **Etiology** may vary to suit the case.

13. Subheads under other headings will vary with material available. (See text treatment of other diseases for suggestions).

14. Omission of heads and subheads is to be made when there are no data to record thereunder.

15. References in the body of the text should usually be inserted at the end of the sentence.

Where one of the references in the bibliography at the end of the paper is to be referred to, enclose in parentheses (the author's name, date of publication, colon and page) thus:—(Jones 1914 : 27). The author's name may be omitted when it is clear, from the context, to which article it is that reference is made.

When an article, not listed in the references given at the end of the paper, is to be cited, the parenthetical insertion must include; (the author's name, name of publication (abbreviated), volume or its equivalent, colon, and pages on which the data are to be found) thus:—(Duggar, Fungous Dis. p. 237, 1909 or Peck, Journ. Myc. **7** : 10–14).

16. Confine the discussion to the disease on the suscept or suscepts specified and on closely related plants.

TERM PAPER SUBJECTS

Alfalfa

Root gall caused by *Urophylctis alfalfæ* (Lagerh.) Magn.
Stem blight caused by *Bacterium medicaginis* (Sack.) EFS.
Yellow leaf blotch caused by *Pyrenopeziza medicaginis* Fckl.
Texas root rot caused by *Phymatotrichum omnivorum* (Shear) Duggar (=*Ozonium omnivorum* Shear).
Root disease caused by *Rhizoctonia crocorum* (Pers.) DC. (=*R. medicaginis* DC.).
Downy mildew caused by *Peronospora trifoliorum* De Bary.

Apple

Black root rot (=Xylaria root rot) caused by *Xylaria* spp.
Crown gall caused by *Bacterium tumefaciens* EFS. and Town.
Scald, no pathogene.
Nectria canker (= European canker) caused by *Nectria galligena* Bres.
Brook's fruit spot caused by *Phoma pomi* Passer (= *Cylindrosporium pomi* Brooks).
Blister canker (= Nail-head canker) caused by *Nummularia discreta* (Schw.) Tuls.
Northwestern anthracnose caused by *Neofabrea malicorticis* (Cord.) Jackson = (*Glœosporium malicorticis* Cordley).
Silver leaf caused by *Stereum purpureum* Fr. (also on plums).
Armillaria root rot caused by *Armillaria mellea* (Fr.) Quel.
Frost injury (freezing).
Pink rot caused by *Cephalothecium roseum* (Fries) Corda.
Sooty smudge (= Sooty blotch, commonly confused with Fly speck) caused by *Glœodes pomigena* Colby.

Soft rot caused by *Penicillium expansum* Link.
Jonathan spot, no pathogene.

Asparagus

Rust caused by *Puccinia asparagi* DC.
Root disease caused by *Rhizoctonia crocorum* (Pers.) DC. = (*R. medicaginis* DC.).

Aster

Yellows, pathogene an unknown virus.
Wilt caused by *Fusarium* sp.

Banana

Wilt (=Panama disease) caused by *Fusarium cubense* EFS.

Barley

Smuts: Loose, caused by Ustilago nuda (Jens.) Kell. and Sw.; covered, caused by *Ustilago hordei* (Pers.) Kell. and Sw.
Stripe caused by *Helminthosporium gramineum* Rabenhorst.
Spot blotch caused by *Helminthosporium sativum* P. K. and B.
Blight caused by *Bacterium translucens* J. J. and R.

Bean

Downy mildew (of lima beans) caused by *Phytophthora phaseoli* Thax.
Rust caused by *Uromyces appendiculatus* (Pers.) Fries.
Dry root rot caused by *Fusarium martii phaseoli* Burkh.
Pod blight caused by *Diaporthe phaseolorum* (C. and E.) Sacc.
Mosaic, pathogene an unknown virus.

Beet (includes Sugar Beet)

Curley leaf (= Curley top), cause an unknown virus.
Phoma leaf spot and heart rot caused by *Phoma betæ* Frank.
Nematode disease caused by *Heterodera schachtii* Schmidt.

Brambles

(Raspberry, Blackberry, Dewberries, etc.)

Anthracnose caused by *Plectodiscella veneta* (Speg.) Burkh. = *Glœosporium venetum* Speg.
Mosaic and leaf curl (yellows), pathogenes an unknown virus.
Double blossom caused by *Fusarium rubi* Wint.
Spur blight caused by *Mycosphærella rubina* (Pk.) Jacz.
Cane blight caused by *Leptosphæria coniothyrium* (Fckl.) Sacc.
Orange rust caused by *Kunkellia nitens* (Schw.) Arth. and *Gymnoconia interstitialis* (Schlecht.) Lagerh. (=*Cæoma nitens* Schw.).
Crown gall caused by *Bacterium tumefaciens* EFS. and Town.
Septoria leaf spot caused by *Mycosphærella rubi* (Westendorp) Roark.

Cabbage

Black leg caused by *Phoma lingam* (Tode) Desm.
Yellows (=Wilt) caused by *Fusarium conglutinans* Wollenw.
Downy mildew caused by *Peronospora parasitica* (Pers.) DeBary.

Cacao

Pod rot and canker caused by *Phytophthora faberi* Maubl.

Carnation (Dianthus)

Anthracnose caused by *Volutella dianthi* Atk.
Bud rot caused by *Sporotrichum poæ* Pk.
Leaf spot caused by *Septoria dianthi* Desm.

Celery

Sclerotinia rot caused by *Sclerotinia sclerotiorum* (Libert) Massee (=*Sclerotinia libertiana* Fckl.).
Early blight caused by *Cercospora apii* Fresnius.

Cherry (sweet and sour)

Leaf blight (=Yellow leaf or Shot hole) caused by *Coccomyces hiemalis* Higg. (=in part *Cylindrosporium padi* Karst.).
Powdery mildew caused by *Podosphæra oxycanthæ* (DC.) DeBy.

Citrus

Scab caused by *Cladosporium citri* Massee.
Melanose, a stem end rot caused by *Phomopsis citri* Fawcett.
Canker caused by *Bacterium citri* (Hasse) Jehle (= *Pseudomonas citri* Hasse).
Wither-tip (Die-back) caused by *Glomerella cingulata* (Stoneman) Spaulding and v. Schr. (=*Colletotrichum glœosporioides* Penz.).
Citrus blast (=Black pit) caused by *Bacterium citriputeale* C.O. Sm.
Nematode disease caused by *Tylenchus semipenetrans* Cobb.
Brown rot and canker or gummosis caused by *Pythiacystis citrophthora* E.H. Sm. and R.E. Sm.

Clover

Anthracnose caused by *Colletotrichum trifolii* Bain (= *G. caulivorum* Kirch).
Stem rot (=Crown rot) caused by *Sclerotinia trifoliorum* Erik.
Eelworm disease caused by *Tylenchus dipsaci* Kühn.
Clover rust caused by *Uromyces fallens* (Desm.) Kern and *Uromyces trifolii* (Hedw.) Lev.
Powdery mildew caused by *Erysiphe polygoni* DC.

Cocoanut

Bud rot caused by *Phytophthora* sp. (*Bacillus coli* (Esch.) Migula?)

Coffee

Leaf spot caused by *Stilbella flavida* (Cke.) Kohl.
Rust caused by *Hemileis vastatrix* Berk. and Broom.
Leaf spot caused by *Cercospora coffeicola* Berk. and Curtis.
Leaf rot caused by *Corticum koleroga* (Cke.) v. Höhn (=*Pellicularia koleroga* Cooke).

Conifers

Brown felt blight caused by *Herpotrichia nigra* Hartig and *Neopeckia coulteri* (Pk.) Sacc. (on spruce and cedar).
Pestalozzia blights and cankers caused by *Pestalozzia hartigii* Tubeuf and *P. funerea* Desm. (spruce, pine, hemlock, fir).
Seedling root rot caused by *Rhizina undulata* Fr. (=*R. inflata* (Schäff) Sacc.).
Shoe string root rot caused by *Armillaria mellea* (Fr.) Quel.
Red rot caused by *Fomes pinicola* Fr.
Stem girdle of seedlings and transplants (=White spot), no pathogene.
Heart rot of Douglas Fir and other conifers caused by *Echinodontium tinctorium* Ell. and Ev.

Corn

Stewart's disease caused by *Bacterium stewartii* EFS.
Ear rot caused by *Diplodia zeæ* (Schw.) Lev.
Root rot caused by *Gibberella saubinetti* (Mont.) Sacc. (and *Fusarium moniliforme* Sheld.?).
Downy mildews caused by *Sclerospora* spp.
Brown spot caused by *Physoderma zeæ-maydis* Shaw.

Cotton

Anthracnose caused by *Glomerella gossypii* (Southw.) Edg. = (*Colletotrichum gossypii* Southw.).
Texas root rot caused by *Phymatotrichum omnivorum* (Shear) Duggar (=*Ozonium omnivorum* Shear).
Southern blight caused by *Sclerotium rolfsii* Sacc.
Ascochyta blight caused by *Ascochyta gossypii* Sydow.
Wilt caused by *Fusarium vasinfectum* Atk.

Cranberry

Scald and blast caused by *Guignardia vaccinii* Shear.
Anthracnose caused by *Glomerella cingulata* (Stoneman) Sp. and von Schr. var. *vaccinii* Shear.

Cucurbits (cucumber, squash, melon)

Bacterial wilt caused by *Bacillus tracheiphilus* EFS.
Mosaic or white pickle, pathogene an unknown virus.
Scab caused by *Cladosporium cucumerium* Ell. and Arth.
Angular leaf spot caused by *Bacterium lachrymans* EFS. and Bryan.
Anthracnose (of melons) caused by *Colletotrichum lagenarium* (Pass.) E. and H.

Downy mildew caused by *Peronoplasmopara cubensis* (B. and C.) Clint.
Powdery mildew caused by *Erysiphe polygoni* DC.

Egg Plant

Leaf blight and fruit rot caused by *Phomopsis vexans* (Sacc. and Sydow) Harter.

Flax

Wilt caused by *Fusarium lini* Bolley.
Heat canker, no pathogene.
Rust caused by *Melampsora lini* (DC.) Tul.
Seedling blight or canker caused by *Colletotrichum linicolum* Pethybridge and Lafferty (= ? *Colletotrichum lini* Bolley).

Grape

Anthracnose caused by *Glœosporium ampelophagum* (Pass.) Sacc. (=*Sphaceloma ampelinum* DeBy.).
Dead arm (=Necrosis and Ripe rot) caused by *Crptosporella viticola* (Red.) Shear (= *Fusicoccum viticolum* Reddick).
Powdery mildew caused by Uncinula necator (Schw.) Burrill (=*Oïdium tuckeri* Berk.).

Hops

Powdery mildew caused by *Sphærotheca humili* (DC.) Burr.

Hyacinths

Wakker's yellow disease caused by *Bacterium hyacinthi* (Wakker) EFS.

Cedar (Incense)

The dry rot caused by *Polyporus amarus* Hedgcock.

Lettuce

Downy mildew caused by *Bremia lactucæ* Reg.
Drop caused by *Sclerotinia sclerotiorum* (Libert) Massee (=*Sclerotinia libertiana* Fckl.) and *S. minor* Jagger.
Gray mold caused by *Botrysis cinerea* Pers.

Maple

Anthracnose caused by *Glœsporium apocryptum* Ell. and Ev.
Decay caused by *Fomes fomentarius* (L.) Fr.
Tar spot caused by *Rhytisma acerinum* (Pers.) Fr.

Melons (see under Cucurbits)

Narcissus

The bulb rot (=Nematode disease) caused by *Tylenchus devastatrix* Kühn.

Oak

Powdery mildews caused by *Microsphæra alni* (Wallr.) Salm.; *Phyllactinia corylea* (Pers.) Karst.; *Erysiphe trina* Hark.
Heart rot caused by *Polyporus sulphureus* Fr.

Oats

Crown rust caused by *Puccinia coronata* Cda.
Blight caused by *Bacterium avenæ* Manns (= *Pseudomonas avenæ* Manns).

Olive

Knot (=Tubercle) caused by *Bacterium savastanoi* EFS.

Onion

Neck rot caused by *Botrytis allii* Munn.
Pink rot caused by *Fusarium malli* Taub.
Mildew (=Blight) caused by *Peronospora schleideni* Ung. (=*P. schleideniana* DeBy.).
Smudge (=Anthracnose, Black spot) caused by *Cleistothecopsis circinans* (Berk.) Stevens (= *Vermicularia circinans* Berk=*Colletotrichum circinans* (Berk) Vogl.)
White rot caused by *Sclerotium cepivorum* Berk.

Peach

Scab caused by *Cladosporium carpophilum* Thüm.
Yellows, pathogene an unknown virus.
Black spot (Bacterial shot hole) caused by *Bacterium pruni* EFS. (also on plums and cherries).
California peach blight caused by *Coryneum beyerinckii* Oud.
Powdery mildew caused by *Sphærotheca pannosa* (Fr.) Lev.
Crown gall caused by *Bacterium tumefaciens* EFS. and Town.
Rosette and little-peach, pathogene an unknown virus.

Pear

Leaf spot caused by *Mycosphærella sentina* (Fr.) Schröt. (=*Septoria pyricola* Desm.).
Scab caused by *Venturia pyrina* Aderh.
Leaf blight caused by *Fabrea maculata* (Lev.) Atk. (=*Entomosporium maculatum* Lev.); also on quince.

Peas

Blight caused by *Mycosphærella pinodes* (Berk. and Blox.) Stone (=*Ascochyta pisi* Lib.)
Streak (sweet peas) caused by *Bacillus lathyri* Mann and Taub.; also on clover.
Powdery mildew caused by *Erysiphe polygoni* DC.

Pecan

Rosette, pathogene unknown.
Scab caused by *Fusicladium effusum* Wint.

Pine

Twig blight caused by *Cenangium ferruginosum* Fr. (=*C. abietis* (Pers.) Rem.).
Mistletoe caused by *Razoumofskya campylopoda* (Engelm.) Piper.
Leaf cast (=Leaf blight, Twig blight) caused by *Hypoderma deformans* Weir and *Hypoderma strobicola* Tubeuf (=*Lophodermium brachysporum* Rostrup).
Red rot caused by *Fomes pinicola* Fries.

Plum

Plum pocket (Bladder plum) caused by *Exoascus pruni* Fckl.
Leaf spot caused by *Coccomyces pruniophoræ* Higg.
Silver leaf caused by *Stereum purpureum* Fr.
Wilt caused by *Lasiodiplodia trifloræ* Higg.

Poplar

Heart rot caused by *Fomes igniarius* (L.) Fries.
Rust caused by *Melampsora medusæ* Thüm.
Cankers caused by *Cytospora chrysosperma* (Pers.) Fr. and *Dothichiza populea* Sacc.

Potato

Hopper burn (Tip burn), cause unknown.
Leak caused by *Pythium* spp., *Rhizopus* spp. and *Mucor* spp.
Black heart, cause not a pathogene.
Black leg caused by *Bacillus atrosepticus* van Hall (See Phytopath. **12** : 444).
Early blight caused by *Alternaria solani* (Ell. and Mar.) Jones and Grout. (=*Macrosporium solani* Ell. and Mar.).
Leaf roll, pathogene an unknown virus.
Common scab caused by *Actinomyces scabies* (Thax.) Güssow (=*Oöspora scabies* Thax. = *Actinomyces chromogenus* Gasp. = *Streptothrix scabies* (Thax.) Cunn.).
Wart caused by *Synchytrium endobioticum* (Shilb.) Percival (=*Chrysophlyctis endobiotica* Schilb.
Bacterial brown rot or wilt caused by *Bacterium solanacearum* EFS. (= *B. nicotina* Ueda).
Wilts caused by *Fusarium oxysporum* Schlect. and *Verticillium alboatrum* Reinke and Berth.
Powdery scab caused by *Spongospora subterranea* (Wallr.) Johns.
Silver scurf caused by *Spondylocladium atrovirens* Hartz.

Rhubarb

Crown rot caused by *Phytophthora cactorum* (Leb. and Cohn) Schröt. and *P. parasitica* var. *rhei*. Godfrey.

Leaf spot caused by *Phyllosticta straminella* Bres.
Anthracnose caused by *Colletotrichum erumpens* Sacc.

Ribes (currants, gooseberries)

Powdery mildew (of gooseberries) caused by *Sphærotheca mors-uvæ* (Schw.) B. and C.
Cane blight caused by *Botryosphæria ribis* Gr. and D.
Mycosphærella leaf spot caused by *Mycosphærella grossulariæ* (Fr.) Lind.
Anthracnose caused by *Pseudopeziza ribis* Kleb.

Rose

Crown gall caused by *Bacterium tumefaciens* EFS. and Town.
Powdery mildew caused by *Sphærotheca pannosa* (Fr.) Lev.
Rust caused by *Phragmidium subcorticium* (Shrank.) Wint.

Rye

Stem smut caused by *Urocystis occulta* (Wallr.) Rabenh.

Snapdragon

Leaf spot caused by *Phyllosticta antirrhini* Sydow.

Sorghum

Smuts caused by *Sphacelotheca sorghi* (Link) Clint., *Sphacelotheca cruenta* (Kühn) Potter and *Sorosporium reilianum* (Kühn) McAlp.

Spinach

Mosaic (Blight), pathogene an unknown virus.

Sugar Cane

Mosaic or stripe, pathogene an unknown virus.

Sweet Potato

Dry rot caused by *Diaporthe batatis* Harter and Field (= *Phoma batatæ* Ell. and Halst.).
Soft rot caused by *Rhizopus nigricans* Ehrenb.
Yellow stem (=Stem wilt or Stem rot) caused by *Fusarium hyperoxysporum* Wollenw., and *Fusarium batatatis* Wollenw.
Scurf caused by *Monilochætes infuscans* E. and H.
Black rot caused by *Sphæronema fimbriatum* (E. and H.) Sacc.

Timothy

Rust caused by *Puccinia phlei-pratensis* Erik. and Henn.
Leaf smut caused by *Ustilago striæformis* (West.) Niessl.

Tobacco

Wild fire caused by *Bacterium tabacum* Wolf and Foster.
Black fire (=Angular leaf spot) caused by *Bacterium angulatum* Fromme and Murray.
Root rot caused by *Thielavia basicola* (B. and Br.) Zopf.
Granville wilt caused by *Bacterium solanacearum* EFS. (= *B. nicotina* Ueda).

Tomato

Winter blight or streak, cause uncertain (? *Bacterium lathryi*).
Fusarium wilt caused by *Fusarium lycopersici* Sacc.
Fruit rot caused by *Phoma destructiva* Plow.
End rot, no pathogene.
Buckey rot caused by *Phytophthora terrestria* Sherb.
Bacterial spot caused by *B. vesicatorum* Doidge (=*Bacterium exitiosum* Gard.).
Leaf mold caused by *Cladosporium fulvum* Cooke.
Downy mildew (=Late blight) caused by *Phytophthora infestans* (Mont.) DeBy.

Trees

Injury to trees by smelter fumes.
Mistletoe caused by *Phorodendron* spp.

Walnut

Bacterial blight caused by *Bacterium juglandis* (Pierce) EFS.

Wheat

Root rot caused by *Helminthosporium sativum* P. K. and B.
Take all caused by *Ophiobolus cariceti* (B. and Br.) Sacc.
Rosette (=So-called take all), cause unknown.
Leaf rust caused by *Puccinia triticina* Erik.
Scab caused by *Gibberella saubinetii* (Mont.) Sacc. (=*Fusarium culmorum* (W.G. Sm.) Sacc.
Flag smut caused by *Urocystis tritici* Koern.
Yellow stripe rust caused by *Puccinia glumarum* (Schm.) E. and H.

NECROTIC DISEASES

FIRE BLIGHT

This is one of the most common and best known bacterial diseases of plants occurring in North America. It also occurs in China, Japan and New Zealand. It commonly affects apples, pears, quinces and occasionally plums, apricots, prunes, cherries and a few ornamental and wild plants related to the apple family (Snow 1922 : 519).

SYMPTOMATOLOGY

The **Morphologic Symptoms** and **Signs** of this disease will be presented in the order in which they manifest themselves during the season on different parts of the tree, beginning with the cankers in the spring.

Hold-over cankers. These are the sources of inoculum for the primary infections in the blossoms in the spring. Study the typical cankers on the limbs of apple and pear trees provided and OBSERVE:—

1. The smooth, more or less sunken area in the bark—the canker, its margin sharply delimited by a definite crack. In cankers in which the pathogene is active this margin is not sharply defined. (See illustration specimens; photographs; Cornell Bul. 272, fig. 16; or Smith 1920, fig. 282.)

2. The margin. Note that it is bounded by a crack formed by the drying away of the diseased tissue from the healthy when the active progress of the pathogene is suddenly checked. Dry or cold weather may thus check the enlargement of the canker. These specimens were collected in the autumn or winter.

3. The surface of the canker. Note that it is smooth, seldom roughened or wrinkled. It is often checked at the margin in drying. Compare with the healthy bark in this respect. Locate the lenticels both in the diseased and in the healthy bark. What is their structure and function?

Make a V-shaped cut across the margin of a canker through to the wood and DETERMINE:—

4. How deeply the disease extends. What tissues are affected? Make a DRAWING of the canker studied. Label fully.

These cankers are formed during the summer and early autumn and in many of them the bacteria pass the winter dormant, or only slightly active in the partially living tissues along the margin. With the increased temperature and the beginning of growth activities in the spring, these bacteria often become active, work rapidly into the adjoining healthy tissue, increase the area of the canker and ooze out through the lenticels to the surface in sticky, milky drops. Study photograph; Cornell Bul. 272, fig. 16; or Smith 1920, fig. 282 and 283. OBSERVE:—

5. That the advancing margin of the canker is not distinctly evident here. The diseased area covers nearly all the surface shown in the illustration, except on the extreme left.

6. The large viscid milky drops oozing out and running down the limb; above to the left, two small globules just oozing out from the lenticels. (Read Cornell Bul. 272 : 40–41; also Ont. Bul. 176 : 15–23.)

Make a DRAWING of this canker from the photograph. Label fully.

Blossom blight. Bees, (?) wasps and flies visit these active cankers in the spring, to feed on the exuding sap and then visit the opening blossoms where they leave behind some of the bacteria which adhered to their feet and mouth parts. Here in the nectar and in the injuries made by the insects' claws in the tender tissues of the flower, the bacteria multiply rapidly, killing the blossoms. From these primary inoculations visiting bees carry the inoculum to other blossoms giving rise to secondary blossom infection. Study the specimens provided; Ontario Bul. 176, frontispiece and figures 8 and 9; Smith 1920, fig. 277; or photograph. OBSERVE:—

7. The dead and blackened flowers. The leaves of the spur are also dead and brown. The bacteria have spread down the pedicles into the spur. These dead and blackened blossom spurs are usually the first striking evidence of the disease in the spring. The oozing cankers are usually overlooked. Cankers are often formed in the branch about the base of the blighted spur.

Make an outline DRAWING of a blighted blossom spur and canker.

Fruit blight. Frequently only one blossom on a spur is infected and by the time the bacteria have killed it and worked their way down the pedicle to the spur itself, the uninfected blossoms have developed fruit of a considerable size. From the spur the bacteria now work into the base of these fruit pedicles and by way of them into the growing fruit. Study Hesler and Whetzel 1917, fig. 90; illustration specimen; or photograph. OBSERVE:—

8. The blackened pedicle and discolored fruit. A fruit affected in this way usually shrivels and drops from the tree. It may cling to the twig as a blackened and shriveled mummy. Note that the leaves of the spur are also dead and shriveling.

9. That in rainy, muggy weather the bacteria ooze from these blighted fruits and blossoms in sticky drops as they do from the hold-over cankers.

10. In early stages of the invasion the discolored and slightly sunken tissues about the base of the stem and extending toward the blossom end. The sticky, milky drops oozing from the diseased area. The large ones on the pedicle.

11. That the cruculio and aphids frequently introduce the bacteria into the fruit through their punctures. The pathogene does not always enter the fruit by way of the pedicel (Cornell Bul. 272, fig. 14).

Make DRAWINGS (from the specimens or photographs) showing these different phases of fruit blight.

Twig blight. The bacteria from the diseased blossoms and fruits are carried by sucking insects, chiefly aphids, to the tips of the growing shoots and water sprouts and are there introduced through the wounds or punctures made by the insects into the tender succulent tissues. Here they multiply rapidly, killing the shoot and causing the form of the disease known as twig blight. Blighted and healthy twigs (fresh or preserved) are provided. Examine and OBSERVE:—

12. The contrast between the diseased and healthy portions of both the twigs and leaves. You may be able to find the dried ooze. See illustration specimen. SKETCH to show contrast between diseased and healthy twigs.

13. That in some of the specimens the dormant buds in the axils of the leaves just below the blighted portion have been prematurely forced. This is a striking case of prolepsis. Explain. See illustration specimen; photograph; or Hesler and Whetzel 1917, fig. 86.

Limb blight. The bacteria often spread extensively in the bark of the limb or branch about the base of a blighted blossom spur, twig or water sprout or from insect punctures into which the bacteria have been introduced. Such limb cankers are commonly limited to a part only of the circumference of the branch and may eventually heal out, the parts above remaining alive and healthy. In other cases the canker completely girdles the branch, the leaves and bark above eventually dying although not invaded by the pathogene. Why?

Leaf blight. The leaves on blighted twigs are usually more or less invaded by the bacteria along the midrib by way of the petioles.

Examine specimens (best fresh) and NOTE:—

14. The diseased midrib and adjacent tissues at first hydropsic then oxidizing to a reddish brown color and drying out. Drops of ooze along petiole and midrib. See Smith 1920, fig. 280 and 285. DRAW.

Leaves may also show lesions (leaf spots) in the blade independent of twig lesions. This type of leaf lesion is common in the west. Examine specimens; or Heald 1915, figs. 1–3 and OBSERVE:—

15. The size, shape, location of the lesions, and their color characters. How do these leaf spots originate? DRAW.

Origin of the cankers. Examine the specimens of cankers again very carefully and observe:—

16. That the cankers almost always surround the base of the spur, twig or water sprout, or that there is at least one or more of these within the area. Sometimes it is a fruit spur which the bacteria have entered by way of the blossoms; more frequently it is a water sprout, the tender succulent growth of which is very favorable for the rapid development of the disease. The bacteria kill the water sprout down to the trunk or limb and spread out into the bark often for a considerable distance around the base of the sprout. See illustration specimens and photograph. If the previous drawing of the canker does not show the dead spur or dead water sprout, DRAW to show it.

Cankers occasionally originate in other ways. Sometimes the pruning knife carried the inoculum. Cankers may also start from insect wounds in the bark (See Ontario Bul. 176 : 40–43).

The **Histologic Symptoms** may best be understood by a comparison of the healthy with the diseased tissues in a twig.

Make thin cross-sections through one-year-old twigs, which have blighted part way down, cutting the sections where the lesion has extended down one side of the twig thus giving diseased and healthy tissues in the same section. (Supplement with prepared slides.) OBSERVE:—

17. The affected area. Which tissues are involved? How recognized? Are all the cells within the diseased region affected?

18. The effect of the pathogene on the epidermis, cork, cortex, sclerenchyma fibers, medullary rays, phlœm, cambium, xylem and pith. Compare with the healthy condition of the cells as to form, size, color and contents.

19. The relatively large cavities scattered throughout the affected portion. How may these be accounted for? (Smith 1920, 365 and figs. 286, 287, and 288.)

20. The stratified appearance just outside of the phlœm. To what due?

21. The bacteria in the tissues. Where located?

DRAWINGS:—(*a*) Make a diagrammatic DRAWING of the cross-section of the diseased twig to show the position of healthy and diseased tissues; (*b*) a much larger and detailed DRAWING of a portion of a cross-section of the affected twig. Make the drawing include both healthy and diseased bark.

Study closely the margin of the lesion next to the healthy tissues. In case the section was taken from a twig in which the pathogene was inactive the diseased and healthy tissues will be separated by a cork layer. If the pathogene was active no cork layer will have yet been formed. DETERMINE:—

22. Which was the condition in the twig from which the section provided was cut.

If young shoots are available make thin cross- or longi-sections near the growing tip of both diseased and healthy. Read Stewart 1913 : 356–361. Examine the sections (free-hand or prepared) from diseased and healthy twigs. MAKE:—

23. A comparative study of the histologic conditions in the two.

24. Compare and contrast your findings with those of Stewart and Bachmann as reported in the above reference.

ETIOLOGY

The organism that causes this disease is *Bacillus amylovorus* (Burrill) Trevisan.

Life History. This pathogene is an obligate parasite. There is no true saprogenesis in its life history. Laboratory studies on its life history, aside from the facts brought out in the above study of symptoms, are largely limited to a short study of the morphology of the bacillus and inoculation experiments. Before proceeding with the following observations the student should have read carefully that portion of the text dealing with the life history of the organism.

If pure cultures are available, examine them carefully and OBSERVE:—

25. The character of the growth on the surface of the solid media. The effect on the bouillon. Why? How long does it take to produce this effect? Determine by inoculating a tube of sterilized bouillon. What does that indicate as to the rapidity of multiplication? Make a mount from a pure culture and before covering introduce a bee's foot or mouth parts. OBSERVE:—

26. The size and motility of the organism and cell unions. Compare it in size with the mouth parts or claw of the bee's foot. It may now be understood how a bee or fly may easily carry thousands of these bacteria.

DRAW the bee's claw or mouth parts and some bacteria beside it. Maintain relative portions.

Study photograph; Smith 1920 fig. 291; or the stained mount under the demonstration microscope and MAKE OUT:—

27. The flagella, their number, length and distribution over the thallus. They are peritrichic. It is this character together with its rod shape and form that places the organism in the genus Bacillus.

If fresh material is available, crush a bit of the most recently affected tissue in water on a slide, cover and try to DEMONSTRATE:—

28. The living bacteria. How do they compare in size, form and abundance with these from the pure culture. Are they motile?

Pathogenicity Studies. If actively growing shoots of apple, pear or quince are available make inoculations by dipping the point of a sterilized needle into a young bouillon culture and prick into the tip of the growing shoot. Examine the inoculated shoots daily and DETERMINE:—

29. The length of the period from the inoculation until the first evidences of infection appear.

30. The rate of progress of the disease down the shoot. Green apples or pears may also be infected by inoculations with pure cultures. Try it on ripe fruits.

Inoculate the blossoms of apples, pears, quinces or strawberries by atomizing bouillon cultures of *B. amylovorus* into them. OBSERVE and RECORD:—

31. The character of the blossom blight symptoms resulting.

32. The length of time between inoculation and the first evidences of infection.

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BEAN BLIGHT

This is the best known bacterial disease of beans. It apparently affects all varieties of *Phaseolus vulgarus L.*, the field and garden bean, as well as the Lima bean, *Phaseolus lunatus L.* The "bacterial wilt" of beans (Hedges 1922) is a very similar disease caused by a closely related pathogene.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of the disease may be exhibited by all the above-ground parts of the suscept, especially by leaves, stems, pods and seed.

On the leaves. Examine leaves showing the usual well developed lesions, OBSERVE:—

1. The location, size, color and other characters of the lesion. It is an indeterminate leaf spot. Compare its appearance on both sides of the leaf.

2. The zonate character of the spots. How do you account for each zone, especially the opaque, green water-soaked zone which shows best when the leaf is held to the light; also the pale, green outermost zone?

DRAW to show these characters and indicate fully in the labeling, the color and appearance.

Leaf lesions in their early stages are sometimes different from these large, striking spots. Examine recently inoculated seedlings or Smith 1920, fig. 213 and 214. OBSERVE:—

3. The minute translucent or water-soaked, angular spots scattered, mosaic-fashion, over the leaflet. Note that they are bounded by the veinlets. DRAW.

The large leaf spot lesions are sometimes formed by the enlargement and fusion of these minute angular spots but more frequently develop from a center with no evidence of a preliminary mosaic-like stage.

On the stems. Examine the stems of the recently inoculated seedlings. OBSERVE:—

4. The minute hydrotic spots similar to those on the leaflets; not extending deeply into the stem.

On the older stems OBSERVE:—

5. That the lesions are in the form of reddish streaks or dashes extending longitudinally. Compare with the stem lesions of the anthracnose. How are they to be distinguished from these? DRAW lesions of both diseases and label to show differences.

Examine specimens labeled "stem-girdle." NOTE:—

6. The deep lesions girdling the old stalk at a node:—the cotyledon node. Its external characters. DRAW.

7. The depth to which the lesion extends. What tissues are chiefly involved. Weakened by this girdling lesion the stems are easily broken over by the wind when heavily loaded with pods. DRAW.

On the pods. Examine the diseased pods and OBSERVE:—

8. The location, size, color and appearance of the lesions. Pod lesions are often red bordered. (See illustration specimens and colored photographs.)

9. Depth to which the lesions penetrate. Determine by cutting across the pod through the lesion.

DRAW to show form and depth of the lesion. Label fully as to color and other characters.

10. That these pod lesions also start as minute water-soaked areas which gradually enlarge to form the characteristic spots. (See Smith 1920, fig. 218, 219 and 220.)

11. That the fully developed pod lesions vary greatly, from definite circular spots to extensive irregular water-soaked blotches often involving a large part of the pod.

Examine as large a number of diseased pods as are available and make SKETCHES to show the different types of pod lesions. Compare the blight spots with anthracnose lesions on the pod. DRAW and label to show differences.

On the seed. Examine diseased seed (dry) and OBSERVE:—

12. The discolored, sometimes shriveled seeds.

13. The character of the discoloration and effect on the seed coat (as shown under hand lens).

14. Some of the seed showing only a slight discoloration about the hilum.

Blight lesions on colored seeds are distinguished with difficulty from those of the anthracnose. Compare seed affected with each.

On seedlings. The planting of blight-infested seed commonly gives diseased seedlings. These may SHOW:—

15. Water-soaked blight lesions on the cotyledons which eventually shrivel and fall. These lesions are not characteristically unlike spots caused by other pathogenes on the cotyledons.

16. A killing of the growing tip leaving the stalk with cotyledons only; a so-called "snake head" effect. (Read Burkholder 1921 : 63.)

17. A wilting of the seedlings due to vascular invasion by the pathogene. (Read Burkholder 1921 : 64 and Hedges 1922.) A wilting or dwarfing of the older plants is not uncommon in the field.

18. That the vascular system of the seedlings is invaded from diseased cotyledons but for some reason the pathogenic effects of this invasion are often delayed until blossoming time or later when a part or all of the plant suddenly wilts. In sone varieties defoliation follows. (See Burkholder 1921 : 64–66 or photograph.)

19. That a dwarfing instead of a wilting may occur. (Read Burkholder 1921 : 66.)

The characteristic **sign** of this disease is the bacterial ooze exuded from the lesions. Examine freshly blighted plants or dried specimens. OBSERVE:—

20. The amber colored viscid drops, often very minute to be found on the surface of pod lesions and on the under surface of leaf lesions (Smith 1920, fig. 219, 221, and 222). Sometimes they appear in the form of a thin, dry incrustation. Show in SKETCHES.

To study the **Histologic Symptoms** of bean blight make free-hand sections (or use prepared slides) through pod lesions or leaf lesions, or study Smith 1920, fig. 223, 224, 225, and 226. OBSERVE:—

21. Cavities filled with masses of the bacteria.

22. Relation of these cavities to stomates. Why?

23. Which tissues are invaded? Does the pathogene enter the cells?

24. Effect on cell contents. Compare with healthy cells of same kind. DRAW.

Study sections cut across vascular-invaded stems from wilted plants. DETERMINE:—

25. Effects on the cell walls of the invaded vessels; on contents and walls of adjacent cells. DRAW.

ETIOLOGY

Life History. The pathogene, *Bacterium phaseoli* Erwin F. Smith is an obligate parasite, normally passing its entire life within the bean. It probably has no true saprogenic phase in its life history.

Primary Cycles are initiated in the spring from diseased seedlings.

Pathogenesis. Soak diseased seed for a time in water until the coat loosens. Peel the seed coat from the seed. Make two mounts, one from the diseased seed coat and one from the diseased cotyledon, chopping and crushing the tissues. OBSERVE:—

26. The minute, short, rod-shaped bacteria to be detected only with the high power. Are they inside or outside the cells? DRAW.

The pathogene on the seed coat or oozing from the lesion on the cotyledon may be splashed by rain drops to the stems of the seedling, or to the young leaves and growing point of the plumule where it enters through stomata causing the stem lesions or "snake head" effect. These are the primary lesions.

The bacteria may also penetrate to the vascular systems of the cotyledons and enter the vascular bundles of the seedlings causing wilting. They may then break forth to form lesions on the stem or leaves from which the oozing inoculum is splashed by rain to other plants nearby and so give rise to primary infections.

27. To what cycle do the wilting symptoms in this disease belong?

Secondary Cycles. Secondary infections are usually far more numerous and therefore are more destructive than the primary. They are the ones most commonly observed. They are repeated frequently throughout the season, initiated at first from the primary and later from earlier secondary infections.

Pathogenesis. The pathogene is carried by insects or rain from the primary lesions to healthy leaves and pods. Examine several of the leaf lesions carefully to see if you can LOCATE:—

28. The point at which the bacteria were introduced. Was the inoculating agent a biting or sucking insect? Was the lesion a result of stomatal invasion?

With a scalpel, chop in a drop of KOH solution (do not crush) a bit of leaf tissue from the outer opaque green zone of the lesion. Cover carefully and under low power OBSERVE:—

29. The bacteria oozing from the cut ends of the veins. Make a DRAWING of a vein with oozing bacteria.

Examine several deep pod lesions, cutting across the pod through the lesion or shell out the beans. The bacteria in the pod invade the seed where with the ripening of the bean, they lie dormant, until the next spring when the planted seed begins to grow. Can evidences of the invasion of the seed beneath the lesion be detected?

Saprogenesis. Although there is little evidence that this pathogene lives and multiplies outside the living tissues of the bean, some investigators hold that it may live over winter in the stems and leaves on the ground (Ontario Bul. 136 : 12). It will, however, grow readily on culture media. Examine the plate cultures and tube cultures and OBSERVE:—

30. The color, form and consistency of both the buried and surface-growing colonies; effect on the bouillon. DRAW and label to indicate color and other characters.

Make a mount from one of the surface colonies and COMPARE:—

31. The bacteria with those studied from the tissues as to size, shape, cell unions and motility. DRAW.

For flagella see Smith (1920, fig. 227). COPY.

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BLACK ROT OF CRUCIFERS

THIS is a very destructive bacterial disease of cabbage and cauliflower. It also affects kale, radishes, rape, kohlrabi, turnips, Chinese cabbage, mustard and other cruciferous suscepts. It is one of the common vascular diseases of plants. It is common in North America, Europe and Japan. It is also reported from the Philippines and probably occurs the world over.

SYMPTOMATOLOGY

While black rot is primarily a necrotic disease it also shows certain hypoplastic symptoms.

The **Morphologic Symptoms** are exhibited mainly in the leaves and by the effects of the disease on the plant as a whole.

In the leaves. Examine the diseased cabbage or cauliflower leaves provided and OBSERVE:—

1. The large irregular dead or dying areas in the blade. The yellowish and finally light brown color of the lesions.
2. The consistency of the dead tissues compared with the healthy.
3. The location of the lesions in the leaf. What does this indicate with respect to the initial point of invasion?
4. That when held to the light the veins in the affected area are black, hence the name. (See Stewart and Harding 1903, pl. I and II; Smith 1920, figs. 84 and 85; or photographs.)

DRAW to show the leaf lesions.

On the plant as a whole. Study the diseased heads provided or Smith 1911, fig. 99; Russell 1898, figs. 1, 8, 9 and 13; or photographs. OBSERVE:—

5. The wilting lower leaves of recently affected plants.
6. That affected plants are often dwarfed and fail to head. Seedlings often wilt and die.
7. The abscission or dropping of the lower leaves, often on one side of the plant only, exposing the prominent leaf scars.
8. In advanced stages of the disease on half mature plants the long bare stalk bearing a loose dwarfed head at the apex.
9. The naked stalks surmounted by the naked solid head in plants affected toward the end of the season.
10. The effect of stump rot (bacterial soft rot) following black rot. The "heart" of the head becomes a soft rotten mass so that the head may be easily lifted away.

Make SKETCHES to show these symptoms.

The **Histologic Symptoms** are exhibited chiefly by the xylem portion of the vascular bundles. Break away a lower leaf from the diseased head or cut across the midrib of a diseased leaf or through the stalk and OBSERVE:—

11. The blackened ends of the vascular bundles. Are all the bundles affected.

Split the midrib or stalk lengthwise and dissect out one of the blackened bundles and DETERMINE:—

12. How far the blackening extends.

13. Whether any other tissues show evidences of injury. DRAW to show these vascular symptoms.

If diseased turnips or kohlrabi are available cut across the root or thickened stalk and lengthwise also or study Smith 1903, pl. 1 and 2; and 1911, fig. 104 and 106. Examine the internal symptoms. OBSERVE:—

14. That the lesions are distributed throughout the fleshy tissues only in the vascular strands.

Make thin cross-sections (or use prepared slides) through a diseased petiole and examine under the microscope. OBSERVE:—

15. The discolored xylem elements (brown in fresh or alcoholic material, red in prepared sections stained with carbol fuchsin).

16. Location of the brown discoloration. (See Smith 1911 : 315–316 and pl. 17.)

17. Effect on the cell walls, pectinosis and decellulosation. What tissues are thus affected? (See Smith 1911 : 314–315.) Does delignification occur? (Read Smith 1903 : 9–11, 15–19.)

18. The formation of cavities,—cytoclasis. What tissues are involved? (See Smith 1911, figs. 110, 115, 117, 118, 120, and 1920, figs. 95, 96 and 97.)

19. Relation of the bacterial masses to cells in the invaded regions. Do your observations confirm Smith (1911 : 314–315 and 1903 : 10–11)?

Make a detailed DRAWING of a cross-section through a diseased vascular bundle to show the points brought out above.

The presence of masses of the bacteria in the xylem vessels and in the cytolcastic cavities is the characteristic sign of this disease.

ETIOLOGY

The causal organism of black rot is *Bacterium campestre* (Pammel) Erwin F. Smith. It is related to the bean blight pathogene, *Bacterium phaseoli* Erwin F. Smith, forming like that bacterium, yellow colonies on agar. It is also monotrichic.

Life History. This pathogene passes the winter either in the débris of its suscepts in the soil or upon the seed.

Primary Cycles usually occur in the seedlings in the seed bed; sometimes however in the plants after they are set in the field.

Pathogenesis. Where infections first appear in the seed bed, the seed are usually the source of inoculum, probably becoming contaminated during threshing. On the oily exterior of the seed the bacteria may remain alive for a year or more.

Examine the agar plates on which contaminated cabbage seeds have been sown. OBSERVE:—

20. The yellow colonies developing about some of the seed. DRAW.

With your needle remove a bit of the colony to a small drop of water on a slide. Cover, examine and MAKE OUT:—

21. The short rod-shaped bacteria swarming in the mount. Their form, size, motility, and cell unions. Study the stained preparations, microphotographs or Smith 1920, fig. 88 and NOTE:—

22. That the organism is a short rod with rounded ends.

23. That the pathogene bears one streptiform polar flagellum. DRAW.

Just how the bacteria on the seed coat find their way into the seedling is not certain, possibly through injured rootlets, but more probably through wounds made by gnawing insects or through the hydathodes. (Read Smith 1911 : 305–309.) Examine cabbage seedlings just coming through the soil. OBSERVE:—

24. That the seed coat is brought up on the tips of the cotyledons. DRAW. They soon fall to the ground from whence splashing rain drops or wandering insects or slugs may transport the pathogene to cotyledons and the young leaves above.

That they often enter through the water pores has been fully established. Examine a cabbage seedling held for some hours under a bell jar. OBSERVE:—

25. The bead-like drops of water along the margins of the leaf each extruded from a hydathode. DRAW. Make thin longi-sections (or use prepared slides) through these water pores. Study their structure. Bacteria finding their way into the extruded water drop at the mouth of the hydathode are carried in through the opening as the drop slowly evaporates and multiplying there soon invade the vascular vessels which end just beneath the opening.

Make a diagrammatic DRAWING to show inoculation and entrance into the suscept. Why do the bacteria not enter through stomata?

When the source of inoculum is infested débris of last year's diseased plants in the soil, the agents of inoculation and the infection courts are the same as in the case of infested seed.

The bacteria having entered the leaf, whether by way of water pores or wounds, soon begin to disorganize the tissues and entering the spiral vessels of the vascular bundle travel downward finally reaching the stalk and by their activities cause the symptoms characteristic of this disease.

Saprogenesis. It is not certainly known whether this pathogene multiplies to any extent in rotting tissues of the suscept in the soil. It probably does not. It grows readily in artificial media as we have seen. If fresh material is available try making isolations in nutrient agar. (The instructor will demonstrate.)

Secondary cycles are initiated in the same way as the primary, especially through the agency of insects and snails, possibly also by wind. Severe epiphytotics often occur. (See Smith 1920, fig. 79.)

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CAROTOVORUS SOFT ROT

THIS is the best known and most common soft rot disease of plants. It affects a great variety of suscepts in each of which it produces more or less characteristic and peculiar symptoms.

SYMPTOMATOLOGY

Since the **Morphologic Symptoms** of this disease vary somewhat with the plants affected it will be found advantageous to study them in several different plants.

In carrots or turnips. It was in the fleshy roots of the carrot that this disease was first carefully studied (Jones 1901). Examine the diseased roots provided and OBSERVE:—

1. The discolored water-soaked appearance of the lesion where it shows on the surface. It is soft.

2. The intact skin of the root over the lesion.

3. The soft mushy character of the rotted tissues. Crush a bit of it between the thumb and finger.

4. The fetid odor of putrefaction.

5. That the rot is largely confined to the root. Cut through the lesion and NOTE:—

6. The variations in color and firmness of the diseased tissue from the center of the rotted mass toward the healthy tissue.

7. That the rotting of the roots in the field is accompanied in its advanced stages by the sudden wilting of the leaves.

DRAW to show these characters.

In cabbage or cauliflower. In these suscepts the soft rot frequently follows the black rot and causes in cabbage the so-called "stump rot." Study the diseased heads provided and OBSERVE:—

8. That the rotting is largely in the base of the fleshy leaves (or in the fleshy head of the cauliflower).

9. That the cabbage head is thus loosened from the stalk from which it may be readily lifted leaving a slimy rotten "stump."

DRAW to show these symptoms.

10. The external character of the lesion especially the color and consistency as compared with the healthy portions.

11. The intact epidermis covering the soft, mushy tissues within.

12. The fetid odor of the decomposing tissues.

Split one of the partially rotted midribs of the leaf or a diseased branch of the cauliflower head. Examine and NOTE:—

13. The differences in color and consistency of the diseased tissues as the healthy tissue is approached.

In iris. This disease is very destructive to the iris both in Europe and America (Hall 1903). Examine the diseased leaves provided and OBSERVE:—

14. The soft rotted basal portion. Crush a bit of it between thumb and finger. If fresh material is available, split a healthy and a diseased leaf and compare as to color and odor.

15. That the epidermis of the diseased portion is more or less intact forming a rather firm covering for the rotten mass within.

16. The upper advancing portion of the lesion, firmer than below but dark and water-soaked in appearance. SKETCH external and internal view.

17. That the tips and margins of some of the leaves rotted at the base are scorched, dying and drying out. How may this be explained?

18. That the most striking field symptoms are the falling over on the ground of the outer affected leaves or the shriveling and browning of the tips. This latter symptom is, however, easily confused with the dying of the tips due to a fungus leaf spot disease common on iris. See demonstration specimen. SKETCH to show field symptoms.

Examine diseased rhizomes if available. Make a similar comparative study and DRAW.

The **Histologic Symptoms** may best be studied in longi-sections (free-hand or prepared) through the advancing margin of a lesion. Examine such sections and OBSERVE:—

19. The kinds of cells and tissues in the healthy portion. Identify them.

20. Condition of organs and other contents of the healthy cells, especially nucleus, chloroplastids and cytoplasm.

21. The condition of the cell structures and contents in the diseased portion. What symptoms do they exhibit?

22. Condition of the cell walls compared with those in the healthy tissues.

23. Just where is the line between diseased and healthy cells? On what characters is your decision based? What tissues are most affected?

24. That the cells are separated from one another in the rotten tissues by the dissolution of the middle lamella; relation of the bacteria to the separating cells?

Make a detailed DRAWING to show the condition in both diseased and healthy cells and tissues, including the region of the advancing lesion.

ETIOLOGY

The pathogene responsible for this soft rot in such a variety of suscepts is *Bacillus carotovorus* Jones. It was first described from carrots.

Life History. The organism doubtless hibernates in suscept débris or in the soil itself.

Primary Cycles are initiated when the particular plant concerned is in the stage of its development most favorable for the attack. Succulent tissue is a requisite for infection. The iris is usually affected very early in the spring while carrots, turnips, cabbage and cauliflower are first attacked in summer or autumn.

Pathogenesis. The source of the primary inoculum is doubtless the soil or rotting tissues of diseased plants in the soil. The bacteria are carried to the roots or rhizomes by snails, insects and probably flowing water. They can enter apparently only through wounds. Once within susceptible tissue with abundant moisture present they multiply rapidly and cause the disintegration of the parenchyma.

Remove a bit of the rotten tissue to a drop of water on a slide, cover and examine for the bacteria. OBSERVE:—

25. Their abundance, and relation to the cells of the suscept. Are they within or without the cells; motile or non-motile; single or in threads? Are spores present? DRAW.

26. The photomicrograph showing the flagellate stage of the pathogene. How many flagella has each bacillus and how are they arranged? DRAW.

Saprogenesis. This organism is doubtless able to multiply in débris from diseased suscepts so that virulent active inoculum is always present in the soil where the pathogene has flourished the previous season. It grows readily on culture media.

Examine the petri dish culture provided. SKETCH and DESCRIBE:—

27. A typical colony as to form, color, consistency and odor. Make a mount from one of the colonies and compare the bacteria with those from the tissues.

Secondary Cycles are initiated from, and developed in the same manner as, the primary cycles.

Pathogenicity Studies. To give one some idea of the rapidity with which the bacteria destroy tissue, a simple inoculation experiment may be made as follows:—

(*a*) Cut three short pieces from healthy leaves of iris, cabbage or cauliflower and three thin slices of roots of carrots or turnips and place them in clean inverted petri dishes on wet filter paper. (*b*) Flame a needle and puncture one piece of each; these constitute the checks. (*c*) Flame a needle and when cool, draw the point through the pure culture and then puncture a second piece of each. (*d*) Flame the needle and when cool, draw the point through the soft, diseased tissue and puncture the remaining pieces. Label carefully and set away until next exercise when they should be examined. RECORD by sketch or description the results of the experiments.

The instructor will demonstrate the method of isolating the pathogene to get it in pure culture.

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ANGULAR LEAF SPOT OF COTTON

This is the common bacterial leaf spot disease of cotton. It also causes necrotic lesions on the other above ground parts of the plant. It appears to be world wide having been reported from most cotton-growing countries.

SYMPTOMATOLOGY

Morphologic Symptoms. The shape and appearance of the lesions vary somewhat on the different organs of the plant.

On the leaves. Examine living (or preserved) leaves showing lesions. Observe:—

1. The small angular spots in the blade. Their relation to the veinlets (Smith 1920, fig. 238–242). On which side of the leaf do they first appear?; average size; character of early stages.
2. The long, narrow lesions on the veins; color.
3. The water-soaked (hydrotic) character of young lesions as compared with the reddish-brown necrotic character of the older spots. The hydrotic zone bordering the brown center.
4. The coalescence of neighboring spots to form large irregular lesions.
5. The viscid creamy exudate or ooze on the lower surface of the lesions, soon drying down to form a yellowish crust; a diagnostic sign of this disease, not always to be observed.

Draw to show leaf symptoms.

6. The yellowing of badly spotted leaves, followed by abscission.

On the bracts. Examine diseased bracts and observe:—

7. The large size of the lesions as compared with those on the leaves (Smith 1920, fig. 239).
8. Their form, size, and color. Compare in these respects with leaf lesions. Draw.

On the bolls. Study the lesions exhibited by diseased bolls and observe:—

9. The circular or oval form of the spots, at first hydrotic, green, becoming black; often coalescing (Smith 1920, fig. 245–246).
10. The sunken, wrinkled definitely limited character of old lesions; size. On what part of the boll are they most commonly found? Why? (See Edgerton 1912 : 17.)
11. Effect on young bolls; abscission, dwarfing on one side, etc.

Open diseased bolls and note:—

12. Depth to which the lesions penetrate and color of the affected tissues.
13. Effect on the lint of the older bolls.

Draw to show the various boll symptoms.

On the stalks and branches. Examine diseased plants showing "black arm." Observe:—

14. The more common location of such lesions.

15. The form, size and color characters.

16. The "healing-out" of stem lesions as the plant matures.

DRAW to show stem lesions.

On the seedlings. Examine affected seedlings and OBSERVE:—

17. The character of the lesions on the cotyledons comparing them with those on the bolls and leaves.

18. The spotting, girdling or streaking of the stems. How may this result in injury to the plant later? (Rolfs, 1915, pl. V.)

19. The dwarfing or stunting effect of severe seedling infection on the later development of the plant (Rolfs, 1915, pl. III and VII).

DRAW to show seedling symptoms.

Read the description of symptoms given by Smith 1920 : 314–317; Rolfs 1915 : 7–11; Faulwetter 1919 : 3–4 and check carefully against your own observations.

Compare the symptoms of this disease with those of the cotton anthracnose. What are the most striking differences between the lesions of the two diseases on the different organs of the suscept?

Histologic Symptoms. This appears to be chiefly a disease of the leaf and stem parenchyma. Make thin cross-sections or study prepared slides through the young leaf and young stem lesions. OBSERVE:—

20. Character of the injury to the cells about a stomatal invasion; effect on cell contents. Are the bacteria in or between the cells? Are the cell walls dissolved.

21. Effect on the palisade and epidermal cells. What necrotic symptoms can you identify?

22. Depth to which injury extends in the stem and character of the injury. Are the vascular tissues affected? If so which cells and how?

Compare your findings with those indicated by Smith 1920, fig. 243 and 261.

Make DRAWINGS to show the histologic symptoms and relation of the pathogene to the cells.

ETIOLOGY

This disease is caused by *Bacterium malvacearum* Erwin F. Smith, a yellow monotrichic schizomycete.

Life History. This pathogene appears to be an obligate parasite continuing its activities only in the tissues of the living cotton plant.

Primary Cycles. These occur apparently only on the seedlings initiated by the bacteria which have presumably overwintered on the lint of the seed. There is some evidence of internal seed infection as in bean blight.

Pathogenesis. Examine lint from the inside of a diseased boll. Make mounts of stained lint and TRY:—

23. To demonstrate the presence of the bacteria on it. DRAW.

Mount a bit of tissue from the lesions on the cotyledons of diseased seedlings and DEMONSTRATE:—

24. The presence of the bacteria in the tissues. How did the pathogene get from the lint onto the cotyledon?

Examine the lesions on the seedling for drops of the yellow sticky ooze. Mount a bit of the ooze in water. OBSERVE:—

25. That it is composed of a mass of bacteria. Are they motile? Note their shape and size.

Secondary Cycles. The ooze on the seedling serves as the inoculum for the secondary cycles which develop on the leaves and bolls of adjacent plants. The agent of dissemination is driving (wind blown) rain drops. (Read Faulwetter Jour. Agr. Res. **10** : 639–648.)

Pathogenesis. Cut out a bit of leaf tissue from the margin of a lesion and chop it up in a drop of water (KOH, if leaf is dry) cover and examine under low power. OBSERVE:—

26. The masses of bacteria oozing from the diseased tissues. Do they come out of the cut ends of veins or from the mesophyl? Can you find them oozing from the stomata? (See Smith 1920, fig. 261.) DRAW.

Although no saprogenic activity is known to occur in nature, this pathogene is easily cultivated in pure culture on agar or other media giving characteristic colonies and reactions (Smith 1920 : 321–333). Examine the agar plate cultures provided and OBSERVE:—

27. The circular pale yellow shining colonies on the surface (lens-shaped when deep in the agar). Make mounts in water and compare with previous mount from ooze, as to shape, size, and motility of the organism.

28. Effect on beef bouillon. Determine by transferring a bit of one of the colonies to a tube of sterile bouillon and examining it daily for a week. Use a flamed transferring loop.

Examine a stained slide under oil immersion or photomicrograph (Smith 1920, fig. 248) and OBSERVE:—

The polar flagellum. Its length compared with the thallus of the bacterium. DRAW.

Pathogenicity Studies. If young cotton plants (6 to 8 weeks old in greenhouse) are available, inoculate with the bouillon culture (See Smith 1920 : 331–332) by, (1) needle pricks, in young stems and pods, (2) spraying or smearing on to the under surface of the leaves, (3) putting a drop into an open blossom.

Watch for and record the first evidences of infection. Compare the lesions thus produced with those studied on material from the field.

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DAMPING-OFF OF SEEDLINGS

The term, "damping-off," is applied to diseases of a type affecting a great variety of seedlings rather than to any one specific disease. It is also applied to the rotting-off of succulent cuttings. Many species of quite different groups of fungi are known to cause damping-off, but as here used the name will be restricted to the diseases produced by certain phycomycetous fungi.

SYMPTOMATOLOGY

The most striking **Morphologic Symptom** and the one which gives to this type of seedling disease the name of damping-off, is the peculiar effect of a necrotic lesion in the stem at or near the surface of the ground. Often the lesion originates just below the surface of the soil. A disintegration of the tissues affected results and the plantlet falls over and dies. Root rot may occur instead of damping-off and may prove fatal except where adventitious roots are put out above the lesion in which case the seedling may recover.

Study the damped-off seedlings provided or photographs. OBSERVE:—

1. The withering of the stem tissues at or near the surface of the soil.

2. The sharp angle made by the fallen stem. While affected plants usually fall over, they may remain erect and wilt if the vascular tissues are sufficiently rigid.

3. The dying of all the plants in affected areas. These damped-off spots are quite characteristic of infested seed beds.

DRAW to show these characteristic symptoms.

Histologic Symptoms. The pathologic effects of these pathogenes are of the simplest sort—a direct killing of the protoplasts—necrosis. Study sections through the advancing margin of the lesion, using fresh material. OBSERVE:—

4. The plasmolyzed and disorganized cell contents.

5. The water-soaked appearance of the diseased tissues due to the diffusion of the cell sap into the intercellular spaces, forcing out the air,—hydrosis.

ETIOLOGY

A rather large number of fungi are now known which produce the above symptoms in seedlings. (See Hartley 1921 : 2–7.) As indicated above, only phycomycetous forms will be here considered. Of these it is one of two species that is usually responsible, either *Pythium debaryanum* Hesse or *Phytophthora cactorum* (Cohn and Lebert) Schroeter (=*P. fagi* Hartig). These two species are very much alike in structure and habits so that the following outline should serve for either.

Life History. These pathogenes exhibit in their life history both primary and secondary cycles, with a short period of pathogenic activity and a well marked saprogenic phase, especially in the case of *P. debaryanum*.

Primary Cycles are initiated at or shortly after the germination of the suscept. Sometimes the seedlings are killed before they get above ground.

Pathogenesis. Both fungi pass their saprogenic existence in the soil, probably always in association with suscept débris. Both produce resting sexual spores,—the oöspores, which carry them through the winter. These germinate and produce the inoculum for the primary cycles. Mount and examine some of the mature oöspores provided (in pure cultures). OBSERVE:—

6. The form, size and color of the sexual bodies.

7. The old oögonial sac, the outer covering enclosing the simple oöspores. The thick smooth wall of the oöspore and its densely granular, oily contents. DRAW.

Lying in the soil near the seedling, these oöspores germinate (in the case of *P. cactorum* at least) by sending forth a germtube at the tip of which is produced one or more conidia. Study Cornell Bul. 363, fig. 18. COPY one or more of the drawings.

These conidia germinate either by swarmspore formation or by germtubes and infect the tender tissues of the seedlings (see Cornell Bul. 363, fig. 16–17). Conidial germination will be studied later.

The mycelium developed from the primary inoculum spreads rapidly in the succulent tissues of the seedlings, secreting enzymes which enable it to penetrate the cellulose walls, and toxines which kill the protoplasm. Study thin longi-sections (freehand or prepared) through diseased stems. OBSERVE:—

8. The relation of the mycelium to the cells of the plant; inter- or intracellular? Are haustoria produced?

9. The structure of the mycelium; densely granular, branched, nonseptate, often knobbed and swollen. The form and structure may often be better made out in mounts from pure culture.

DRAW showing structure of the mycelium and its relation to the tissues of the suscept.

From this mycelium is sent forth from the diseased tissues short fruiting branches,—the conidiophores, on which are developed conidia just like those produced from the oöspore. Make mounts from diseased stems or from pure cultures showing conidia. Study and OBSERVE:—

10. Their peculiar form, apical papillæ and coarsely granular contents.

11. Their attachment to the conidiophore at their larger end. DRAW showing conidiophores with conidia.

These conidia function as the inoculum for the secondary cycles. In *P. debaryanum* it is held that mycelium spreading through the soil from seedling to seedling usually functions to initiate the secondary infections. This may also be true at times for *P. cactorum.*

Saprogenesis. The mycelium in the dead tissues continues to grow and produce conidia so long as the food supply is not exhausted. As the nutrients begin to fail there develops on the mycelium within the tissues the sexual bodies, which eventually mature into oöspores. Tease apart in a drop of water bits of diseased stems which contain oöspores or use mounts from pure cultures provided. OBSERVE:—

12. The large globose bodies, –the oögonia, and the smaller closely appressed antheridia.

13. The manner in which they are borne on the mycelium and their positional relation to each other.

14. Their thin, hyalin walls and granular contents.

15. The aggregation in some, of the denser protoplasm in the center,—the oösphere, and the surrounding layer of hyalin protoplasm,—the periplasm.

16. The impregnation tube penetrating from the antheridium, through the oögonium wall and periplasm to the oösphere. What is the function of this tube?

Make DRAWINGS showing the structure of the sexual bodies and fertilization of the oösphere.

The fertilized oöspores now go into a resting period and will not germinate at once. Normally this dormant period extends through the winter. During the spring, changes in the oily protoplasm of the oöspore take place which make its germination possible. With germination of these oöspores (already studied) the primary cycle is completed.

Secondary Cycles are initiated repeatedly during the growing-season as has already been pointed out, either by conidia or by mycelium spreading through the soil. If viable conidia are available study conidial germination; or Cornell Bul. 363; fig. 16–17; and read p. 97–99.

Make DRAWINGS illustrating ways in which conidia (sporangia) germinate; also the structure and germination of the swarmspores. How do the germtubes gain entrance to the suscept tissues? Why can they not penetrate mature tissues?

The further developments of the pathogene during the secondary cycles duplicate those of the primary.

Pathogenicity Studies. The rapid destructiveness of these pathogenes in a bed or flat of closely crowded seedlings is strikingly brought out by a simple inoculation experiment.

Under the direction of the instructor each student may inoculate the seedlings provided, with pure cultures of one or the other of these fungi. Keep under moist conditions and examine daily for first evidences of infection and successive symptoms. Make notes on conditions under which the inoculated plants were kept, length of incubation period, and effect of removing plants to dry conditions before all go down, and other facts observed.

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LATE BLIGHT OF POTATOES

This is the most destructive disease of the potato in the humid temperate regions of the world. It is especially severe in northeastern United States. Tomatoes are also affected in some regions.

SYMPTOMATOLOGY

During damp, muggy weather in August and September, circular areas in the fields are often to be observed in which all the tops of the potatoes are dead or dying. (See N. Y. (Geneva) Bul. 241, pl. 7 or photographs.)

The **Morphologic Symptoms** of this disease are very characteristic and seldom to be confused with those of any other disease of the potato.

On the leaves. Examine the material provided (leaves and photographs) and OBSERVE:—

1. The large spots or lesions with indefinite margins. Their position in the leaflets.

2. On holding the leaf to the light, the dark water-soaked or opaque appearance of the diseased areas.

3. On the lower surface of the lesion, a white frost-like mildew about the margins of the diseased spots;—the distinctive **sign** of this disease (see illustration specimen, photograph; or N. Y. (Geneva) Bul. 241, pl. 11). In dry weather this mildew may be difficult to see. Why?

4. That the lower leaves are usually the first attacked. They die and shrivel on the stem. (See illustration specimen; or N. Y. (Geneva) Bul. 241, pl. 10.)

Compare these leaf spot symptoms with those of the Alternaria (early) blight as shown in the material and photographs provided.

Show in DRAWINGS the differences in the leaf symptoms of these two diseases. Show both sides of the spots.

On the tubers. The disease may also occur on tubers, causing a reddish brown rot. (Read Connecticut Ann. Rept. **1904** : 364, and see pl. 33; also photographs.)

Study the material provided and OBSERVE:—

5. The sunken spots on the surface of the diseased tubers. To what due?

6. The color and consistency of the diseased tissues shown on cutting the tuber. How deep do the lesions extend?

DRAW to show tuber symptoms as observed in specimens and photographs.

This disease is also destructive to the tomato in some sections of the United States. If material is available study the symptoms on this suscept and show in DRAWINGS. (See California Bul. 175 : 9–15 and fig. 3–5; also Virginia Bul. 192 : 7–10 and fig. 5–8.)

ETIOLOGY

One of the phycomycetous fungi, *Phytophthora infestans* (Montagne) DeBary, is the cause of this disease. Most species of Phytophthora are virulent and destructive pathogenes.

Life History. It is generally held this pathogene remains in constant association with the living suscept throughout its life history. There is, however accumulating evidence to indicate that it has a saprogenic phase of importance.

The **Primary Cycle** originates from inoculum produced on diseased shoots from tubers infected the previous season. That they also originate from overwintered oöspores in the soil is probable.

Pathogenesis. The fungus passes the winter as mycelium in diseased tubers. Make thin tangential sections of the diseased tubers, stain with eosin and OBSERVE:—

7. The mycelial threads between the cells. Their densely granular contents. Can you demonstrate the presence of haustoria? (Jones et al. 1912 : 30, pl. 8. fig. 53.) What is their function?

8. The general absence of septa in the mycelium; this fungus is a cœnocyte. Toxines secreted by this mycelium kill the adjacent cells of the tuber and their soluble contents are set free for the pathogene. DRAW to show the relation of the mycelium to the cells of the suscept.

The mycelium in the diseased tuber grows out into one or more of the shoots resulting in a systemic invasion. The diseased shoot is weak and dwarfed. On such shoots conidia are produced in great abundance. Such plants become centers of dissemination, i. e., sources of inoculum, for the primary infections. After these conidia are produced and dispersed the fungus in these systemically invaded shoots dies with the suscept. (Read Melhus 1915 : 99–100, and see pl. 5–7.)

Make a DIAGRAMMATIC DRAWING to show the origin of the primary inoculum. The primary infections occur on the leaves of nearby stalks, are relatively few and serve to afford inoculum for the secondary cycles. The lesions and pathogene structures are like those resulting from the secondary infections in the leaves and will be examined in detail below.

The **Secondary Cycles** arise from the conidia produced on the primary lesions. These conidia are distributed by wind during rainy or foggy weather, to nearby healthy plants where they germinate.

Pathogenesis. Make mounts of these conidia from material provided (freshly blighted leaves or cultures, if available) and OBSERVE:—

9. The shape, contents and color of conidia. These conidia upon germination act either as sporangia, giving rise to swarmspores or as spores producing germtubes directly.

Germinate fresh spores, if available; or see N. Y. (Geneva) Bul. 241, pl. 12; California Bul. 175, fig. 8; or photographs. MAKE OUT:—

10. The manner of germination; number and form of swarmspores and their motility by means of flagella.

DRAW to show points observed or COPY from plates or photographs studied.

After swimming about for a short time in the drop of rain or dew on the leaf, the swarmspores come to rest, round up, invest themselves with a thin cellulose wall and germinate by a germtube. Study slides, figures in bulletins, or photographs. OBSERVE:—

11. The relative size of the germinating swarmspore and the sporangium. The difference in form of the active and germinating swarmspores.

12. The two methods of entrance of the swarmspore germtube into the leaf. COPY to show this.

This germtube gives rise to mycelium within the leaf tissues like that observed in the tuber. Haustorium-like branches are often sent from the intercellular mycelium into the leaf cells. From this mycelium branches are put forth on the underside of the leaf to form the conidiophores.

Scrape some of these conidiophores from the leaf and mount in water. OBSERVE:—

13. Their form and structure. Distinguish the conidiophores from the large leaf hairs. Note the peculiar swellings on the branches.

14. The ovate conidia; their manner of formation and their attachment to the conidiophores. (See also photograph or N. Y. (Geneva) Bul. 241, pl. 12.)

If freshly diseased leaves are available, make sections through the margin of the spot. Mount and study, MAKING OUT:—

15. How the conidiophores emerge from the tissues of the leaf. (See also N. Y. (Geneva) Bul. 241, pl. 12, fig. 2; Maine Bul. 169, fig. 15 or California Bul. 175, fig. 7.)

Make a DRAWING to show the leaf surface, conidiophores emerging and conidia attached both at the apex and sides of the branch.

These conidia in turn give rise to other secondary infections, on healthy leaves or they fall to the ground during rains and produce swarmspores in the soil. These reach the growing tubers which they penetrate by a germtube This gives rise therein to the mycelium that is to carry the pathogene over to the next season, invade the shoots and develop conidia for the primary infections as we have already seen.

Saprogenesis. As already indicated, a true saprogenic development of this pathogene in nature is not fully established. Murphy (Scientific Proc. Roy. Dublin Soc. n. s. **16** : 462–464. 1922) has shown that the conidia may live for many days in the soil often producing secondary conidia. The tubers may thus become inoculated from contaminated soil long after the tops have been killed. No true saprophytic growth however appears to occur as he found no production of mycelium by these soil-infesting conidia. He also reports (in a letter Oct. 27, 1921) the discovery of oöspores on the surface of diseased tubers.

That these oöspores may remain in the soil over winter and produce inoculum (conidia) for primary infections in the spring as long ago claimed by W. G. Smith (Diseases of Field and Garden Crops 295–310) is thus highly probable. This is rather to be expected since most other species of Phytophthora are known to produce them often in great abundance. Moreover

P. infestans in artificial culture will sometimes produce oöspores (Jones 1912 : 57–69 and Clinton 1911 : 753–774. pl. 38–40).

Of what importance is it to determine with certainty whether or not *Phytophthora infestans* may pass the winter in the soil or in suscept débris?

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SCLEROTINIA ROT

This is a very common disease of a great variety of plants. It is especially destructive in rainy seasons or in very moist situations. Due to the different symptoms exhibited by its great variety of suscepts it has come to be known under several names as; stem rot in seedlings and herbaceous plants, soft rot in stored roots and bulbs, and "drop" in lettuce.

SYMPTOMATOLOGY

Since the **Morphologic Symptoms** of this disease often vary strikingly among the many different kinds of plants affected, all cannot be presented in this exercise. Our study will be restricted to some of those commonly exhibited by herbaceous ornamentals and vegetables.

The most usual point of injury is the base of the stem, but the leaves of such plants as cabbage and lettuce, especially when packed together in heads are commonly affected. Fleshy roots, when stored together often suffer from this disease.

Stem rot. The name stem rot is applied to the disease in those cases where the lesions are chiefly at the base of the stem. The stalks of many annuals and herbaceous perennials such as peonies, sunflower, bachelor's button and hollyhock, are often attacked near the base causing a crown rot. In very young seedlings it often produces a damping-off effect. These symptoms are in general much the same on the different suscepts. Examine the diseased stems provided. Observe:—

1. A bleaching of the normal color of the tissues accompanied by softening and hydrosis. If fresh material is available, examine for these characters.

Where the air is sufficiently humid for the rapid development of the disease certain characteristic **signs** soon appear. Examine diseased specimens which have been kept for a time in a moist-chamber. Observe:—

2. The densely matted pure white felt of mycelium on the surface of the lesion.

3. The large black sclerotia embedded in the mycelial mat. These sclerotia are often formed within hollow or pithy stems. (See photographs and demonstration specimens.)

4. The creamy white color of the young sclerotia commonly covered with large clear drops of a watery exudate.

Make sketches to show these signs and symptoms of stem rot.

Drop. This term is applied especially to the effect produced by the disease on lettuce. Examine the diseased plants provided and observe:—

5. The collapsed and wilting state of the plant. It has "dropped." Sketch a diseased and healthy plant.

6. The point of injury and extent of the lesion.

7. The soft slimy condition of the affected tissues. Note also odor, color, and consistency of the affected parts. How does this rot compare with the carotovorus soft rot?

The typical **signs** of the disease are usually to be observed in affected **lettuce heads.** OBSERVE:

8. The loose white felty masses of mycelium, on and between the decomposed leaves. Large knots of compact aggregations of mycelium which are the beginnings of sclerotia.

9. In somewhat older material the mature sclerotia which have become hard with black, tough outer walls.

Root rot. This symptom is exhibited chiefly by fleshy roots and tubers like those of the carrot and turnip. This is common in storage cellars and in packages in transportation. Bulbs are rarely or never affected by this disease.

Examine the diseased roots provided and OBSERVE:—

10. The characteristic **signs** of the disease; (*a*) the felty white mycelium covering most of the surfaces of the lesion; and (*b*) the hard tuber-like sclerotia in various stages of development. The sclerotia are never formed inside the tissues of fleshy roots as they are in the pith of stems. Why?

11. The hydrotic zone in the tissues about the mycelium-coated central portion of the lesion. Note that it is rather firm.

12. The soft decay of the tissues beneath the mycelial felt.

DRAW to show these external signs and symptoms.

Cut the root lengthwise through the lesion. OBSERVE:—

13. The depth to which the lesion extends. What tissues of the root are involved?

14. Differences in color, texture, taste and odor of the diseased tissues compared with those of the healthy.

DRAW to show these internal symptoms.

To study the **Histologic Symptoms** mount a bit of the diseased tissues from just back of the advancing margin of the lesion. DETERMINE:—

15. The effects of the pathogene on the cells of the root. What necrotic symptoms can you detect. DRAW to show your findings.

This mycelium secretes a toxine which kills the cells of the suscept. When the sclerotia are forming, large drops of liquid are extruded upon their surfaces. This also contains the toxine.

The mycelium also secretes an enzyme which dissolves the middle lamella from between the cells.

16. Make thin sections of the healthy portion of the root and mount in a large drop of the liquid from a bouillon culture. Mount similar sections in water. Cover in a petri-dish to prevent evaporation. After an hour or so, EXAMINE:—

17. Compare the two mounts as to changes in the tissues. RECORD the results in the form of notes or sketches.

ETIOLOGY

The disease is caused by the ascomycete *Sclerotinia sclerotiorum* (Libert) Massee* (=*Sclerotinia libertiana* Fuckel)

The **Life History** of this pathogene varies somewhat with the suscepts affected. Its main features are the same in all however.

The **Primary Cycle** is usually initiated in the spring, in northern climates.

Pathogenesis. The source of inoculum for the primary cycle is the overwintered sclerotium. Examine the specimen provided. OBSERVE:—

18. The large black sclerotium with the trumpet-shaped apothecium; its stalk and concave disk. DRAW.

From this apothecium are shot forth the ascospores which constitute the inoculum of the primary cycle. The general nature of this inoculum may be determined as followss: Gently break the seal and remove the cover from the glass preparation dish in which are living apothecia. Watching sharply, OBSERVE:—

19. The tiny cloud of spores that puff from the disk of the apothecium. (See illustration photograph.) This cloud is composed of ascospores which caught by the breeze are borne about and lodging in a suitable infection court initiate the cycle. All of the ascospores of an apothecium are not discharged at one time.

For a microscopic study of these ascospores and the method of their production examine a crush mount of a bit of the cup or better thin longi-sections, (free hand and prepared). OBSERVE:—

20. The layer of sack-like asci forming the inner surface of the cup and containing the ascospores,—the hymenium.

21. The slender thread-like paraphyses among the asci.

22. The dense interwoven mass of mycelial hyphæ upon which the ascus layer rests and from which the asci arise—the hypothecium.

23. The remaining more loosely interwoven portion constituting the bulk of the apothecium,—the excipulum.

Make an outline DRAWING of a median section of an apothecium to show the location of these parts.

With the high power, MAKE OUT:—

24. The shape of the ascus and its attachment to the hyphæ of the hypothecium. The pore at its apex through which the ascospores are discharged. This is closed with a plug which is dissolved out when the spores are ripe. This plug stains blue with iodine. Demonstrate it.

25. The ascospores; number in an ascus, size, shape, septation.

Make a large detailed DRAWING showing several asci, paraphyses and ascospores.

Once within the infection court where sufficient moisture and some nutrients are available, these ascospores germinate and proceed to infect the suscept.

To study ascospore germination, invert an apothecium by attaching its stipe with a bit of gummed paper to the inside of the lid of a deep petri dish in the bottom of which a drop of water on a clean slide has been placed. After a few minutes remove the slide and replace with another. Examine the first slide to make sure that ascospores have been discharged into the drop, then place it in a moist-chamber (inverted petri dish) and examine it after an hour or so from time to time. (Potato agar in a petri dish may be substituted for the drop of water on the slide.) DETERMINE:—

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* See Wakefield, Phytopath. **14** : 126–127. 1924.

26. From what place on the spore the germtube arises; number of tubes from each spore, relative size of spore and germtube, branching, contents. DRAW. This germtube branches, forming the mycelium which spreads through the tissues, causing the rot.

Remove a bit of the rotten tissue and tease apart in a drop of water on a slide. Cover and examine for mycelium. OBSERVE:—

27. Size, septation, branching, color, relation to suscept cells, (inter- or intracellular?). Do haustoria occur?

As this pathogene grows readily in culture, the mycelium may be advantageously studied in the agar culture in the petri dish provided. Carefully cut out a small square from the advancing margin of the colony, transfer to a slide in a drop of water, cover and with little or no crushing, study the mycelium, comparing this with that found in the tissues.

DRAW to show the characters of the mycelium.

As the mycelium kills the tissues, it rapidly extracts and appropriates the food substance therein. As this food supply begins to dwindle the mycelium forms gnarls of short intertwining hyphæ at different points in or on the lesion. These rapidly enlarge and mature forming the large black sclerotia.

Make thin sections of one of the sclerotia and OBSERVE:—

28. Its structure; (*a*) the black outer layer or rind made up of about two or three layers of cells whose walls are thick and brown to black; (*b*) the inner white medulla made up of densely interwoven thick walled hyphæ. These sclerotia are storage organs and the food substance stored is largely in the thickened walls of these medullary hyphæ.

DRAW to show the structure of rind and medulla much enlarged. (See DeBary, Morph. and Biol. Fungi, p. 30–32, fig. 14.)

Asexual spores, known as micro-conidia are often produced in abundance especially from the mycelium as it grows old and from the germtubes of ascospores produced in a non-nutrient media like water. Search for them in the mounts of ascospores germinating in water and in mounts from old cultures provided. OBSERVE:—

29. Their minute size and globose form; the form of the conidiophores upon which they are borne. Note that they are produced in chains. DRAW.

The microconidia of this species have never been observed to germinate but those of the related species *Botrytis cinerea* have been germinated by Brierley (Kew Bull. Misc. Inf. 1918 : 129–146). There is considerable circumstantial evidence indicating that the microconidia of *S. sclerotiorum* may function as inoculum for secondary cycles.

Saprogenesis. With the death of the tissues and the exhaustion of the nutrients therein by the pathogene the mycelium finally dies. There is no evidence that it increases or even lives for any length of time in the decaying débris of the suscept or in the soil.

The sclerotia alone remain alive to carry the pathogene through the dry season or the winter, lying dormant in the soil until favorable conditions of temperature and moisture combined cause it to produce the apothecia with their ascospores. The sclerotia appear to require about a month of dormancy after they mature before they will respond to conditions favorable to germination and in many cases several months of rest seem to be necessary.

Secondary Cycles. Since the apothecia normally appear in the spring (or in the tropics in the wet season) the frequently to be observed appearance of new lesions during the summer may possibly be the result of microconidial infection.

The mycelium may also spread over the soil from diseased to neighboring healthy plants in which case the mycelium itself serves as the inoculum for secondary infection.

Pathogenicity Studies. The pathogenicity of *S. sclerotiorum* and the development of the symptoms of the disease may be easily studied as follows:—

30. Inoculate the base of stems of beans, sunflower or lettuce with an agar block containing mycelium (from petri dish cultures). Cover inoculum with moist cotton and wrap with strip of tinfoil.

31. Inoculate roots of carrot or turnip by placing inoculum on the surface (injured or uninjured), cover with a bit of wet cotton and place the roots in a moist chamber.

32. If living ascospores are available, inoculate some potato plants with a suspension of these in water. (See Pethybridge, 1911 : 431.)

KEEP NOTES on method of inoculation, character of infection courts, length of incubation period and development of symptoms.

Reisolate the pathogene from the lesions appearing on inoculated plants.

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BOTRYTIS BLIGHT OF PEONIES

THIS is a common and very important disease of the peony. There appear to be in fact two Botrytis blights so similar in symptoms as to be distinguished only by the pathogene structures themselves.

SYMPTOMATOLOGY

The **Morphologic Symptoms** and **Signs** of the Botrytis blights of the peony are so similar that for the purposes of this study they are to be considered as identical. All organs of the plant may be affected.

On young shoots. Study photographs and specimens provided. OBSERVE:—

1. That when the plants first come up in the spring, shoots from three to ten inches high suddenly begin to wilt and fall to the ground.

2. At the base of these wilting shoots, the brown rotten lesion usually involving the entire circumference of the stem and extending some distance above the surface of the soil (through sometimes entirely below ground). The wilting noted above is due to this basal stem rot.

3. Covering the surface of some of these lesions the greyish brown felty mat of conidiophores of the pathogene, appressed or broken away in the dry specimens; very evident in fresh specimens.

4. On some of the lesions in their more advanced stages, sclerotia, small black bodies the size of a pin-head embedded in the cortical tissues. These are usually found at the base of the stem where it was covered by the soil. These sclerotia constitute the diagnostic signs of the disease.

5. That the entire shoot soon withers and dries. SKETCH to show the symptoms in blighted young shoots.

As the season advances shoots continue to wilt from lesions at their base. However, the proportion of such cases decreases rapidly as the stem tissues mature and harden. Toward the middle of the season the stems attacked at the base wilt but do not fall over. The development of the lesion is much slower and the wilting is often preceded by a yellowing or a purpling of the foliage.

Study such specimens and photographs as are available showing the characters of the stem rot in the older shoots.

On the buds. The bud rot is the next symptom of the disease to develop after the stem rot of the young shoots. The symptoms exhibited by affected buds are of two general types; a "bud blast" of the small immature buds when they are from the size of a pea to half an inch in diameter and a "bud rot" which is the name applied to the disease as it manifests itself in affected buds after they are nearly or quite mature, just before blooming.

Bud blast. Examine the material available. OBSERVE:—

6. The black or brown undeveloped small buds in the cluster. It is usually the first or leader bud of the shoot that is affected. Note that some

of the secondary buds are larger and healthy. They grow rapidly after the death of the main bud.

7. The brown felt of conidiophores with grey conidia. This is often wanting or difficult to detect with the naked eye especially in dry specimens.

SKETCH to show bud blast.

Bud rot. Examine one of the large buds provided. OBSERVE:—

8. The general discoloration of the floral parts. The bud is killed. It fails to open.

9. The felt of conidiophores, usually most noticeable about the base of the bud where it is attached to the stem.

10. That the lesion often extends down the stem for some distance below the bud. Study illustration specimens and colored photographs.

SKETCH to show the external characters of bud rot.

Some of the dry rotted buds have been moistened to put them in a more characteristic condition. Carefully dissect one of these and OBSERVE:—

11. That the innermost parts of the bud are involved, the petals being very soft and decayed.

12. That the petals do not separate readily from one another being held together by the mycelium of the pathogene. By carefully tearing apart two adjoining petals the stretched and breaking hyphæ may sometimes be detected.

13. Mats or wefts of mycelium which may sometimes be observed in the cavities of the bud.

The bud rot phase of this disease is probably its most serious feature. Why?

On the blossoms. (Blossom rot.) Sometimes the half open buds are attacked. They turn brown and collapse and become a rotted mass. This occurs only during an epiphytotic of the disease in very wet weather.

Examine the dried specimens provided. They give only a partial idea of this condition as it appears in the field. Compare with healthy blossoms as shown in the colored photograph. SKETCH.

On the upper stem. It has already been seen that the stem at the base of the bud may and usually does become involved. Examine specimens that show upper stem lesions. OBSERVE:—

14. The brown discoloration and shrunken character of the diseased stem.

15. In many cases, very characteristic alternating light and dark bands or zonations especially marked on stems just below rotted buds. (See colored photographs and specimens.)

16. On some of the lesions, conidiophore felts. These are usually wanting on stem lesions.

17. Occasionally stem lesions showing the sclerotia of the pathogene as small black bodies bursting through the epidermis. Examine illustration specimens or photographs showing these.

18. That sometimes the pathogene spreads from blasted small secondary buds or from blighted leaves into the stem, forming canker-like lesions. Examine illustration specimens and photographs.

SKETCH to show different types of upper stem lesions.

On the leaves. Leaf lesions while they may occasionally appear early in the season become characteristic and abundant usually at the time of, or just after, the blossoming period. Examine specimens and photographs provided. OBSERVE:—

19. The location of the majority of the lesions, i. e., the part of the blade involved.

20. The average size and the color characters of the lesions. Note evidences of zonation in some of them similar to those on the stem.

21. Conidiophores. Where most abundant? That they are more scattered, i. e., less felty than on shoots and buds.

SKETCH to show both surfaces of leaf lesions.

On the root. The crowns and roots may also be affected, resulting in a "brown rot" of the roots. If specimens are available study, NOTING:—

22. The firm more or less dry brown decay of the tissue. The entire diameter of the root is involved.

The **Histologic Symptoms** are those usually to be observed in necrotic tissues, plasmolysis, granulation, oxidation, discoloration of cell walls and cell contents, grumosis and the like. These can only be studied to advantage in fresh material.

ETIOLOGY

There are at least two species of Botrytis which cause the blighting of peonies. The small sclerotial species is *Botrytis pæoniæ* Oudemans. The large sclerotial species is *Botrytis* sp. of the *cinerea* type. The *Botrytis pæoniæ* Oudemans appears to be the more common and destructive and is the one here considered.

Life History. This fungus exhibits in its life history both primary and secondary cycles with characteristic pathogenic and saprogenic phases.

The **Primary Cycles** are initiated and completed, chiefly at least, on the young shoots which become diseased shortly after they come up in the spring.

Pathogenesis. So far as is known conidia alone constitute the primary inoculum. These are produced in early spring from overwintered mycelium in the old diseased stubble or stem débris on the ground, also from germinating sclerotia in the old stems or in the surface soil.

Diseased stems have been brought in from the field and placed in moist chambers. Examine the material and OBSERVE:—

23. The numerous tufts of brown conidiophores covered with grey conidia,—the primary inoculum. Do they arise directly from mycelium or from sclerotia?

SKETCH to show these conidial clusters.

Mount some of the conidia and OBSERVE:—

24. Their form, size and color. These conidia are carried by the rain, to the young shoots that are coming up nearby. Here they lodge and if conditions of moisture and temperature are favorable they germinate and penetrate the tender young tissues. Germinate conidia and study them. DRAW.

With penetration by the germtube, infection occurs, the first evidence of injury showing in two or three days. The mycelium penetrates the tender tissues in all directions killing them presumably by means of toxines which it secretes.

Make thin sections of diseased stems provided and STUDY:—

25. The morphology of the mycelium and its relation to the tissues of the suscept. Is it inter- or intracellular? DRAW.

From the mycelium, conidiophores are soon thrust forth to the exterior and conidia quickly and abundantly produced. Make mounts of conidiophores from diseased stems. OBSERVE:—

26. Their forms, structure and the way in which conidia are borne. DRAW.

Saprogenesis. After the shoot dies the mycelium begins to form sclerotia at the surface of the lesion, especially below the surface of the soil, where moisture conditions are more favorable.

Make thin sections across a sclerotium. Mount and study under the microscope. OBSERVE:—

27. Its structure; the outer black rind cells and the central white medulla. DRAW.

The diseased shoots shrivel and fall to the ground, harboring the sclerotia until next season. The mycelium frequently spreads down from the diseased shoots into the crowns and roots causing root rot.

Secondary Cycles. The conidia produced on the diseased young shoots are carried chiefly by ants to the developing buds where in the sugary exudate they quickly germinate, penetrate and cause the bud blast.

Other secondary cycles are exhibited later in the bud rot, blossom rot, upper stem rot, and leaf blight. The fungous structures produced, their relation to, and effects upon the tissues are the same as those of the primary cycle. Sclerotia are rarely produced, however, in any of the suscept organs other than the stem though they are occasionally to be found on or in rotted buds. The mycelium of the secondary cycles in old leaves and especially stems give rise in the moist days of spring to great numbers of conidia which may also initiate primary cycles.

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BROWN ROT OF STONE FRUITS

This is primarily a disease of plums, peaches and cherries. Apples, pears and quinces in America are sometimes affected, but the brown rot of pome fruits in Europe is caused by another, though closely related pathogene. The brown color of the affected tissues has given to the disease the name of brown rot. In the case of certain varieties of apples, affected fruits finally become jet black.

SYMPTOMATOLOGY

The most common **Morphologic Symptom** of this disease is the rot of the fruit, but the disease also affects the blossom, twigs and limbs at times. Leaf spotting rarely occurs.

Blossom blight. This form of the disease is the first symptom to appear on peach trees if the spring weather is at all favorable. It sometimes occurs also on plum and cherry blossoms (Brooks and Fisher 1924 : 2–3, pl. 1). Examine the specimens provided and OBSERVE:—

1. Effect of the disease on the blossoms; similar to the effect of the fire blight on apple blossoms.
2. The greyish brown spore tufts of the fungus on the pedicels and young fruits.
3. The drop of gum at the base of the pedicel. Sometimes the entire blossom is embedded in this exudate. Gummosis is a common symptom of several diseases of the peach and other stone fruits.
4. The little canker or girdle at the base of the pedicel (Smith 1891, pl. 5, fig. 1–3 or photographs).
5. The grey brown spododochia of the pathogene on the brown petals or calyx cups and on the cankers;—these are the characteristic **signs** of the disease.

DRAW to show blossom blight symptoms.

Twig blight. The girdling of the twig commonly causes the dying of the parts above. Twig blight seems to occur only on peaches and plums. Examine the specimens provided and OBSERVE:—

6. The dead and shriveled brown leaves of the upper part of the twig; in appearance not unlike the effect of fire blight on apple or pear twigs.
7. The old blossom embedded in a drop of gum at the point where the twig was girdled; or—
8. The old mummified fruit or fruit pedicel where the disease has extended into the bark.

DRAW to show the twig blight form of the brown rot.

Cankers. Brown rot cankers are very common on large and small limbs of peach trees in some peach growing sections. They originate usually from the blighted blossom or fruit spurs. (See Jehle, 1913 : 107). Examine the cankered limbs provided and OBSERVE:—

9. The rough open cankers with evidences of repeated callus formation. What does this indicate as to the age of the canker?

10. Evidences of the diseased twig or spur about which the canker developed.

11. Cankers breaking out above or below the original lesion.

DRAW to show the character of brown rot cankers.

Fruit rot. While green fruits, especially of peaches and plums, often show the rot it is far more common on the ripe fruit. Diseased fruits of peaches, plums and cherries have been provided. Examine these and OBSERVE:—

12. The brown discoloration of the diseased area. The margin of the spot, distinct or indefinite? (See colored photographs.)

13. That at first the diseased tissues do not collapse, the diseased portions retaining their original plump character. Compare this with a fruit in an advanced stage of decay. To what is the difference due?

DRAW to show fruits in early and advanced stages of the rot.

With the finger TEST:—

14. The relative firmness of the diseased and healthy portions of the fruit; strength and character of the epidermis.

Cut into the fresh fruit and NOTE:—

15. The effect of the disease on the tissues as to discoloration, firmness, taste and odor.

The disease is not uncommon on apples and pears, especially fallen fruits. The pathogene usually enters these fruits through insect punctures or other injuries. Examine the fresh fruits provided and COMPARE:—

16. The external symptoms here with those on stone fruits.

17. The internal symptoms exposed to view, on cutting through the lesion, with the healthy tissues.

18. The taste of the diseased flesh with that of the healthy.

Mummification. The fruit is often attacked and killed before it matures and for this reason clings to the tree, instead of falling as is the case with ripe or prematurely ripened fruit. Such diseased fruits dry and wrinkle forming the so-called mummies of brown rot. In the material provided OBSERVE:—

19. The dry and shriveled condition of the fruit; color.

20. Often a cluster of the mummies clinging together, bound to each other by threads of mycelium where they touch.

21. The sporodochia of the fungus crowded or scattered over the surface; color?

SKETCH a mummied plum or peach.

Examine the mummied apples provided. OBSERVE:—

22. That the mummies are black in marked contrast to the brown mummies of the peach. (Plum mummies are sometimes blue black.) Only certain varieties of apples appear to produce black mummies when attacked by the brown rot fungus. The black color is confined to the skin. The flesh within is brown. (See Heald 1906 : 82–90. pl. 1.)

23. That the sporodochia of the fungus are not produced on such mummies except at cuts or wounds.

The **Histologic Symptoms** of this disease are chiefly a discoloration of the protoplasm of the affected cells followed by plasmolysis and collapse. Is there dissolution of the cell walls? (See Cooley 1914 : 302–304 and compare Valleau 1915 : 384–385, 391.)

To study these effects on the tissues of the suscept cut thin sections from a diseased plum or apple through the advancing margin of the lesion and compare the cells in the healthy and in the diseased portions. RECORD your findings in drawings and notes.

ETIOLOGY

The pathogene causing the brown rot of stone fruits is *Sclerotinia cinerea* (Bonorden) Woronin. A closely related species *Sclerotinia fructigena* (Persoon) Schröter, is the common cause of brown rot of apples, pears and other pome fruits in Europe and was long supposed to be the cause of the rot of stone fruits in America. These fungi belong to that group of the ascomycetous fungi known as the Pezizales which are characterized by having an open cup-shaped apothecium in which the asci are borne. In the conidial stage the pathogene has been commonly known as *Monilia cinerea* Bonorden.

Life History. This fungus has a more complicated life history than most pathogenes causing diseases of the more common crops. This is due to the fact that its mycelium may live over winter producing conidia in spring which initiate primary cycles.

Primary Cycles may be initiated from more than one source and by two sorts of inocula, as brought out in (*a*) and (*b*) below.

Pathogenesis. (*a*) The mummies which fall to the ground give rise in the spring to apothecia which discharge myriads of ascospores about blossoming time. The apothecia are very short-lived, appearing over a period of not more than two or three weeks. (See Brooks and Fisher 1924 : 3, pl. 1–2.) Specimens have been provided. OBSERVE:—

24. The goblet-shaped fruit body,—the apothecium with a long slender stipe. Upon what does the length of the stipe depend?

25. The size and color of the apothecium. The upper surface (bearing asci) is when fully expanded, usually flat or even convex with recurving margins. They retain almost their natural color in alcohol.

26. The attachment of the stipe to the mummy; upper or lower side? Why?

Make a diagrammatic DRAWING showing the anatomy of the apothecium; its relation to the mummy and to the surface of the soil.

Crush a bit of one of the apothecia especially provided for this purpose and OBSERVE:—

27. The minute ovate ascospores,—the inoculum. Some of them are still in the asci in which they are formed. DRAW. They germinate by a simple germtube.

(*b*) The mummies which cling to the trees through the winter or the cankers on the limbs harbor the pathogene as living mycelium or as thick-walled conidia. The warm spring rains cause the formation of multitudes of thin walled conidia which it is believed may also function as primary inoculum.

Mummies have been brought into the laboratory and placed for several days in a moist chamber.

Examine one of the mummies and NOTE:—

28. The greyish brown powdery spore tufts. Make an enlarged DRAWING of a part of a fruit showing these sporodochia as they appear under the hand lens.

With the point of a scalpel remove a bit of the spore mass to a slide, cover and examine. OBSERVE:—

29. The rather large hyaline globose or oval bodies scattered through the mount,—the conidia. Variations in form, size, and thickness of the walls.

30. The granular, sometimes alveolate character of the protoplasm of the spore. (See also under the oil immersion demonstration microscope.)

The ascospores or conidia are carried to the opening blossoms, where if the weather be rainy they quickly germinate and the germtubes penetrate the calyx or exposed corolla. The blossom blight form of the disease is thus initiated.

Put some of the conidia in drops of water on slides to germinate. After some hours examine and OBSERVE:—

31. The long septate germtubes. Simple or branched? How many from each conidium? DRAW.

The mycelium spreads throughout the blossom and on protruding conidiophores forms masses of greyish brown conidia,—the inoculum for secondary cycles. From the blighted blossom the mycelium follows down through the fruit spur to the twig or branch killing the bark and causing twig blight or young cankers. In the cankers on the limbs the fungus may reamin alive throughout the winter and provide, as indicated above, conidial inoculum for the primary cycles the next year.

The primary cycles may include a **Saprogenic Phase** when the mycelium lives over in twig cankers or when they develop on fruits. The latter is probably rare as the primary inocula are largely dispersed before the fruits reach any considerable size.

The **Secondary Cycles** are in all probability initiated from conidia developed on the blighted blossoms or from the early secondary infections on fruits and twigs. The blighted twigs, blossoms and green fruit continue to harbor and multiply the parasite during the early part of the season until the maturing fruit offers opportunity for its very general spread and destructive work.

Pathogenesis. Diseased specimens of ripe fruits showing the pathogene are provided (or student may inoculate under direction of instructor). With forceps, remove a bit of tissue from a badly rotted fruit. Tease apart in a drop of water on a slide; cover and study under the microscope. OBSERVE:—

32. The form, structure, contents, septation, branching, size and color of the mycelium. Note variation in diameter of the mycelial threads. (Staining with eosin or methylene blue will bring out the mycelium.) Are the cells penetrated? If available, study the mycelium growing in agar.

DRAW to show the mycelium in the fruit and its relation to the cells.

This mycelium soon masses at certain points beneath the skin of the fruit and bursting through pushes forth a cushion of short branched conidiophores, forming the sporodochium. Make thin sections through these sporodochia, and the skin of the fruit (or use prepared slides), study and OBSERVE:—

33. The cluster of conidiophores protruding through the ruptured epidermis. The cuticle of the fruit pushed back on either side.

34. The densely packed mass of mycelial cells from which the conidiophores arise,—the stroma. Note its pseudoparenchymatous structure.

To study the conidiophores more easily make a mount from the culture or the inoculated fruit. OBSERVE:—

35. Their form and mode of branching, length and diameter, color, contents and septation.

36. The conidia; their mode of formation,—moniliform. It was from this character that the conidial stage first received the name Monilia.

Make a careful DRAWING of the section through the sporodochium to show its structure and relation to the suscept.

From the diseased fruits, especially if attacked when green, the mycelium may penetrate through the cortex of the fruit pedicel to the bark of the twig and cause twig blight. Then passing on down to the larger limbs, it spreads into the bark and initiates the large limb cankers.

Saprogenesis. Many of the rotted or mummified fruits fall to the ground in the autumn or cling to the tree. Those on the tree may, as already seen, harbor the parasite throughout the winter. Those which fall to the ground become more or less buried in the soil and in them just beneath the cuticle in the epidermal regions is formed a crust-like black stroma. Make sections (or use those provided) through the skin of one of these fallen black mummies. Study and OBSERVE:—

37. The black thick-walled cells of the outer layer of the stroma. Their relation to the old dead tissue of the suscept.

38. The thickness of the stroma and character of its inner margin.

DRAW to show structure of stroma and relation to the fruit tissues.

From this stroma there arise in the spring the apothecia, the external features of which have already been studied. Examine a thin section through the apothecium and OBSERVE:—

39. The densely packed hyphæ making up the greater portion of the section,—the excipulum. This is composed of much septate interwoven threads of the mycelium. It forms the bulk of the apothecium and serves as a protection and foundation for the layer of asci that lines the cup,—the hymenium.

40. The make-up of the hymenium; the long slender tube-like asci containing the ascospores. How many ascospores in each ascus? Their form and structure; the thread-like paraphyses standing up between the asci; septate?

The paraphyses at first form the hymenium but as the apothecium expands the asci are formed and push up between the paraphyses and become a part of the hymenium. Make out the pore at the apex of the ascus. (This may be demonstrated by treating a crushed mount with iodine solution. The pore will stain blue.) Supplement your observations with the prepared slides.

Make a DRAWING to illustrate fully the structure of the apothecium.

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BEAN ANTHRACNOSE

THIS is one of the most serious diseases with which the farmer and vegetable gardner have to contend. It occurs wherever beans are grown and, under conditions favorable for the fungus, will ruin an entire crop.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of bean anthracnose may appear on all parts of the suscept especially cotyledons, stems, leaves and pods. Roots are rarely affected (Barrus 1921 : 108.) The lesions are all necrotic.

On the seedlings. OBSERVE:—

1. The elongated, blackened lesions on the stems, with the centers shrunken and in some cases the tissues cracked open.

2. The lesions on the cotyledons, more or less circular, depressed and canker-like (Barrus 1921, fig. 13). Where do the lesions first appear, on the stem or on the cotyledon? Why?

Make a DRAWING of the diseased seedling.

On the leaves. OBSERVE:—

3. The browned or blackened streaks on the under side. The areas between the veins are seldom injured. On the young growing leaves the veins are often killed while the tissues between still continue to grow, so that the leaf becomes distorted (Barrus 1921, fig. 11). Do the lesions show on the upper side of the leaf? DRAW.

On the pods. OBSERVE:—

4. The distinct regularly outlined, much depressed spots; their shape and size.

5. The margin of each, usually bordered with a red zone. Where two spots are near together, the red zones coalesce.

6. The blackened center and, occasionally in the larger lesions, the tissues so shrunken that they are cracked open. Are there any spots lacking the dark centers?

The characteristic **Signs** of the disease are most evident in the pod lesions. Exame these lesions (fresh specimens are best) under hand lens or binocular and OBSERVE:—

7. The minute pimple-like bodies, often arranged in concentric circles on the dark central area of the spot—the acervuli of the pathogene. In fresh specimens these are pale pink due to the oozing mass of spores. In preserved specimens they are pale dirty white in color.

Make DRAWINGS of several lesions on a pod to show the above points.

On the seeds. OBSERVE:—

8. The browned or blackened areas. Do they resemble in any way those on the pods? Are they depressed? Is the seed coat ruptured at any point in the lesion? Are acervuli present? DRAW.

Compare the symptoms of this disease with those of bean blight, shown in the illustration materials.

The **Histologic Symptoms** of the anthracnose are those usual in necrotic lesions; chloroanemia, grumosis, discoloration of cell walls and cytoclasis being easily demonstrated. Make thin cross-sections through a lesion on stem or pod and make out such of these as you can. DRAW.

ETIOLOGY

The pathogene of bean anthracnose is a fungus, *Colletotrichum lindemuthianum* (Saccardo and Magnus) Briosi and Cavara. It is known only in its conidial stage. It is doubtless an ascomycete belonging to the genus Glomerella (Barrus 1921 : 113) as are the related species in which the sexual stage is known. This pathogene was discovered in 1875 by a German mycologist, Lindemuth, for whom it was named.

Its **Life History** is a simple one. But one kind of spores are produced and the secondary cycles are like the primary except that they develop on the pods, stems and leaves of the older plants rather than on seedlings.

The **Primary Cycle** is initiated by conidia from the lesions on the cotyledons. When diseased beans are planted they contain the mycelium of the pathogene, which at the germination of the seed also begins to grow and is ready to produce spores when the cotyledons push above the surface of the soil.

Pathogenesis. Scrape some material from a lesion on the cotyledon of a seedling provided. Mount and examine. OBSERVE:—

9. The numerous small hyaline conidia mixed with the débris of diseased tissues. Their shape, size and color. DRAW.

These constitute the primary inoculum. They are washed or splashed by rain to the stalks and first leaves of the seedling which thus inoculated show within a few days the lesions of the primary infection. The cotyledons soon shrivel and fall carrying with them the source of primary inoculum. If dry weather prevails from the time the beans come up until the cotyledons fall, few or no primary infections occur and a clean crop is assured.

The conidia, washed from the cotyledon lesions, germinate on the stem or leaves by a germtube. This penetrates directly into the tissues or forms a resting cell at its tip,—an appressorium (Barrus 1921, pl. 3). Study germinating conidia and OBSERVE:—

10. The number and positional origin of the germtubes. Note that the identity of the conidium is sometimes nearly or quite lost in the developing mycelium.

11. The darker thick walled appressoria at the ends of some of the germtubes. What is their function?

DRAW germinating conidia.

From the surface of the appressorium next to the leaf arises a very slender penetration tube which forces its way through the cuticle into the epidermal cell. Here it enlarges and elongates into the mycelium which branches and spreads through the tissues.

To study this mycelium make thin cross-sections through a lesion on the stem (or pod). Stain with eosin for ten to thirty minutes. Wash with water, cover and study under high power. OBSERVE:—

12. Relation of mycelium to the cells of the suscept; inter- or intracellular?

13. Whether large or small in diameter in comparison with the cells of the bean tissues; septate or non-septate, color (Barrus 1921, pl. 2). DRAW.

There are soon developed in the primary lesions on the seedling leaves and stems, minute fruit bodies. Examine the lesions and OBSERVE:—

14. The number and location of these fruit bodies,—the acervuli. Color? Some of them may show (in fresh specimens) the pink mass of conidia oozing out on the surface of the lesion.

Mount and examine some of the conidia from a primary lesion to see if they are the same as those from the cotyledons. Conidia from the primary lesions are produced for a considerable period and initiate the secondary cycles.

Saprogenesis. The mycelium in the old primary lesions may continue to live and spread saprophytically into the dead tissues of the leaves and stems, on the débris of which in the spring it may produce conidia. These conidia probably seldom function in initiating primary infections. The sexual stage may develop from the saprophytic mycelium in old stems and leaves but is not certainly known for this pathogene.

Secondary Cycles develop on the later leaves and on the pods. The same phenomena of inoculation and infection are exhibited as in the primary cycle.

Pathogenesis. The lesions on pods afford the best material for a study of the structure of the fruit bodies of the pathogene.

Study cross-sections through a lesion. OBSERVE:—

15. The saucer-shaped acervuli, at the surface of the lesion; their relation to the tissues of the suscept; especially the epidermis.

16. The thin mat of mycelium forming the base of the acervulus, from which arise the conidiophores.

17. The size and shape of the spores; the manner in which they are attached to the conidiophores. What are the bubble-like bodies found embedded in the granular contents of each? Do the spores have septa?

18. The dark tapering septate threads which extend above the conidiophores and have somewhat the appearance of spines. These are the setæ. Their function is unknown.

Make a diagrammatic DRAWING of a section through a lesion showing the relation of the acervuli to the suscept tissues. Make an enlarged DRAWING of a portion of an acervulus, showing its structure.

If fresh pods showing the oozing spores are not available examine the cultures provided and OBSERVE:—

19. The pink masses of spores embedded in a gelatinous matrix. It is in this manner that they ooze from the acervulus and are later washed or splashed by the rain to initiate new infections. They are formed so fast by successive constrictions of the conidiophores that many millions may be formed on one badly diseased pod.

If conditions are favorable, the mycelium in the lesions on the pods grows through the tissue and into the seed below it. It is in this manner that the bean seed becomes invaded by the fungus hyphæ which produce spores on the growing cotyledons the following season.

Cut through a bean pod lesion directly over a seed and OBSERVE:—

20. The depth of the diseased area and the relation which the pod lesion bears to the discolored area on the seed. Make a DRAWING showing this relation.

In this manner the pathogene usually completes the cycle without going through a saprogenic period of existence.

Saprogenesis. The mycelium in the pods and stalks develops saprophytically after they are harvested or fall to the ground. Here are produced conidia wherever sufficient moisture and favorable temperature allows. If such débris is scattered with manure on land to be planted to beans, conidia produced thereon may cause primary infections in the spring. Observation indicates that this rarely occurs and that diseased seed is the usual source of the primary inoculum.

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ANTHRACNOSE OF SYCAMORES AND OAKS

This anthracnose is a serious disease especially in the northeastern part of the United States where it is frequently epiphytotic. It affects most of the species of the genus Plantanus, in Europe as well as in the United States. The sycamores are more commonly and destructively affected than are the oaks. However, in certain regions some species of oak esepcially the white oak suffer severely.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of this disease are exhibited by leaves, twigs and small branches. In addition to the characteristic lesions produced on these organs, there are certain general symptoms by which the diseased trees may be detected at a distance.

General symptoms. In the photographs provided, OBSERVE:—

1. The dead, leafless twigs.

2. The irregularity of the direction taken by smaller branches. These twisted and deformed branches are characteristic of this disease.

3. The excessive twigging or fasciculation on the smaller limbs, producing clumps of branches,—witches'-brooms. Note that many of the branches of the broom are dead. Make a DRAWING of the broom-like growth.

In the case of trees badly affected, the foliage becomes thin due to the falling of many leaves, and those remaining appear scorched owing to the large dead areas in them. In the early spring when the leaves are developing, all the leaves on a twig may wither and die due to the death of the twigs. When this type of injury is general it is often mistaken for frost injury.

On the leaves of sycamores. In the material provided, OBSERVE:—

4. The large brown areas in the leaves.

5. That usually the lesion is located along one of the larger veins of the leaf.

These lesions, when first noticeable, are narrow dead areas paralleling the veins on both sides. These dead areas then rapidly enlarge as the tissues of the vein are killed and the portion of the lamina dependent on the vein for water dies and turns brown. Why do most of the lesions extend to the margin of the leaf?

6. The lesions on the petioles.

DRAW a sycamore leaf showing these symptoms.

On the leaves of oaks. In the material provided, OBSERVE:—

7. That in some cases the lesions are similar to those on sycamore leaves in that the dead areas are located along the veins but that more commonly numerous small lesions occur without any relation to the veins. How can the difference in size between the lesions not located in the blade and those located on the main veins be accounted for?

8. That some of the diseased leaves are curled and distorted. How do you account for this?

DRAW to show the symptoms exhibited by oak leaves.

The **Signs** of the disease are the minute fruit bodies of the pathogene. Examine the leaf lesions (oak or sycamore) under the hand lens or binocular and OBSERVE:—

9. The pimple-like fruit bodies,—acervuli, scattered over the lesion; shape, color and size; often most evident on the veins and petioles.

DRAW to show appearance of acervuli on the lesions.

On the twigs. Twigs from oak or sycamore, collected in the early spring, are provided. OBSERVE:—

10. That the twigs are (or were) still alive and that the buds formed the previous autumn have reached maturity.

11. The fruiting stage of the pathogene formed under the cork layer, raising it up in pimple-like pustules. Later the cork layer is ruptured and the fruit bodies are exposed.

12. The terminal portion of the twig is partially or entirely invaded by the pathogene, as indicated by the distribution of the fruit bodies noted above.

Twigs thus affected may put out leaves in the spring which, however, usually wither and die before they reach full size. This is due to the rapid advance of the pathogene in the twigs, killing them.

On the branches of sycamores. In the specimens provided, OBSERVE:—

13. The small diseased areas around the base of the twigs,—cankers.

14. Larger cankers with remains of old twigs at the center showing the origin of the canker.

15. The cortex of the bark raised up and broken through by the fruit bodies of the pathogene. In some cases these fruit bodies are gone and only a cavity is left in the bark.

16. Older cankers in which the dead bark has fallen away and the wood is left bare.

17. That callusing takes place rapidly in some cases and the open wound is covered.

18. That swellings of irregular shapes are often formed at points where witches'-brooms originate.

ETIOLOGY

The anthracnose of sycamores and oaks is caused by the ascomycetous fungus, *Gnomonia veneta* (Saccardo and Spegazzini) Klebahn. Previous to the discovery of the ascigerous stage of the pathogene it was known by several names because of the various types of asexual fruit bodies which the fungus forms. The commonest of the names for the imperfect form is *Glœosporium nervisequum* (Fuckel) Saccardo, this being the name for the acervulus stage which occurs on the lesions on the leaf blades. On dead leaves and petioles a pycnidial form is produced,—The Sporonema stage, and on the twigs, a pseudo-pycnidial form,—the Myxosporium stage.

Life History. The polymorphic character of the asexual fruit bodies found on the different parts of the suscepts which are attacked, makes the life history of this pathogene complicated. Their identity was established by Klebahn in 1905. He also connected the polymorphic conidial stages with the ascosporic stage which is developed on overwintered leaves on the ground.

Primary Cycles may be initiated by several sorts of inocula. No definite investigations have been made to determine the relative importance of these in nature. The possible primary inocula may be: ascospores from overwintered leaves, conidia from the Myxosporium stage on twigs and cankers, and conidia from the Sporonema stage on overwintered leaves. It seems most probable that the primary inoculum in the spring is the conidia from the twigs and cankers or the ascospores in the overwintered leaves.

Pathogenesis. The fungus passes the winter in the living twigs. In autumn, asexual fruit bodies (pseudopycnidia) begin their development on the affected twigs and reach maturity early in the spring. This is the so-called Myxosporium stage.

Examine with the hand lens the twigs with the mature Myxosporium stage. Make freehand sections at right angles to the twig. Stain with eosin, mount and OBSERVE:—

19. That the top of the fruit body is open. Has it a definite ostiolum?
20. The abundance of conidia.
21. The size, shape and color of the conidia.

These conidia are produced in great abundance and ooze out of the fruit bodies in creamy drops or strings. In the presence of abundant moisture they may be washed or splashed to the young unfolding leaves. It seems that the conidia thus disseminated in early spring from the fruit bodies in the twigs are the most efficient of the various possible primary inocula. The other types of primary inocula undoubtedly play some role and must be considered.

In the overwintered leaves, specimens of which are provided, NOTE:—

22. The small pimple-like bodies on the blade or on the petioles. This is the so-called Sporonema stage. Remove one of these fruit bodies to a slide, mount and crush. OBSERVE:—

23. The abundance of conidia coming from the pustules.
24. The remains of the walls which enclosed the spores.
25. The size, shape and color of the conidia. Compare with the conidia obtained from fruit bodies on the twigs.

Make DRAWINGS of this stage.

The pycnidia-like fruit bodies thus formed on overwintered leaves come about by growth of the vegetative hyphæ around the margin of the acervulus, arching up over the conidia until they are enclosed. DRAW.

The conidia in the Sporonema pustules on dead leaves remain viable through the winter and may cause primary infection in the spring if they reach growing parts of the suscept. This may occur through splashing rain.

In the same overwintered leaves showing the Sporonema stage, search for the perithecia. NOTE:—

26. Small black beaks protruding from ruptured places in the leaf epidermis.
27. On the opposite side of the leaf, that a distinct bulge in the surface is evident.

Cut out small bits of the leaves containing perithecia. Mount in water and tease away the leaf tissue. NOTE:—

28. The shape of the perithecium with its beak. What is the function of the beak? DRAW:—

29. Upon crushing the perithecium, the contents.

30. Shape and size of asci; thickness of their walls.

31. Shape and size of ascospores; number in each ascus; septate or non-septate?

Make DRAWINGS of the perithecium, asci and ascospores. (If material is not sufficient see Edgerton, 1908 : 379, fig. 3–7 and pl. 11, fig. 18–23.)

The ascospores are probably forcibly ejected from the perithecium as most ascospores are, and thus they may readily be borne by air-currents to the unfolding leaves and produce primary infection in the spring. That primary infection is commonly initiated by ascospores is evidenced by the fact that leaves on the lower branches are usually the first to show the disease in the spring.

It is thus clear that the primary inoculum may be either conidia from twigs and overwintered leaves, or ascospores from the leaves upon the ground. Falling upon the young unfolding leaves, the conidium or ascospore as the case may be, germinates if moisture be present, sending forth a germtube which may or may not form a secondary kind of spore at its tip—an appressorium. (See Edgerton, 1908; pl. 11. fig. 26–28.) From the tip of the germtube or from the appressorium arises a slender penetration tube which pierces the wall of the epidermal cell on which it lies. Once inside the cell this tube enlarges to form the mycelium. The protoplasm from the germtube or appressorium flows through the penetration tube into the forming mycelium which grows rapidly, branching and spreading through the tissues. A lesion soon appears, as a minute water-soaked spot.

As the lesion enlarges the acervulus type of fruit body is formed. In the prepared sections of acervuli, OBSERVE:—

32. The aggregation of hyphæ radiating from the base of the fruit body.

33. The conidia cut off one after the other from the ends of the conidiophores. These form so abundantly that they pile up into balls or strings and are held together while dry by a gelatinous matrix.

34. The individual conidia; their shape and size. Compare with conidia from Myxosporium and Sporonema stages.

Make DRAWINGS to show the structure of an acervulus in cross-section, including a portion of the adjacent leaf tissue and the vegetative hyphæ.

Saprogenesis. When the leaves fall to the ground the mycelium spreads rapidly to all parts of the leaf and lives saprophytically forming more acervuli and, in moist situations, the pycnidia of the Sporonema stage. The perithecia begin their development after the mycelium has existed saprophytically for a time. The ascospores are mature by early spring.

Secondary Cycles originate from the conidia developed in acervuli on the leaf lesions and petiole lesions. It is suggested by some that the mycelium, extending down from the diseased petioles into the twigs, plays an important part in the infection of the current year's twigs. This has not been proven, but it happens either in this way or by infection during the summer resulting from conidia. The infected twigs are not killed but develop normal buds which put out leaves the following spring. However, the mycelium in the twigs gains enough headway during the autumn and early spring to cause the death of the twigs at a time when the leaves are only partially developed.

References

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BLACK ROT OF GRAPES

The black rot of grapes is of American origin. It was known as a destructive malady in our vineyards for nearly a century before its appearance in Europe about 1885. It is markedly epiphytotic in its occurrence on cultivated grapes.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of black rot may appear on any of the green parts of the vine, including the young canes. The woody parts never show new lesions of the disease.

Leaf spot. The lesions on the leaves are small, more or less circular, necrotic spots. They appear first as blanched areas caused by the disintegration of the chlorophyll in the affected tissues. Study the materal (specimens and photographs provided) and OBSERVE:—

1. The size, color and distribution of the mature lesions on the leaf.

2. The shape of single spots and their relation to the finer veinlets. Study the margin closely; even or crenulate?

3. That the size of the individual spots appears to be affected by the number per unit area of leaf surface. How? Why?

Fruit rot. The first evidences of the lesions on the berry is like that on the leaf, a blanching of the green color resulting in an indefinitely delimited greyish scald-like spot which rapidly changes to a distinct lesion with a brown border. Study the different stages of lesion development as shown by the diseased berries and photographs provided, and OBSERVE:—

4. The three rather definite stages in the development of the lesion; the early grey scald-like phase; the brown rapidly enlarging rot lesion; and the black shriveled mummy stage characteristic of the final effects of the disease.

5. That there is often a concentric zoning of the surface in the brown rot stage. To what due?

6. That the blackening of the lesion sets in usually before the brown rot stage has involved the entire berry. Where does the blackening begin?

Cankers. On mid ribs, canes, petioles and tendrils the lesions take the form of small cankers. Examine the specimens and photographs provided or see Reddick, 1911, fig. 132 and OBSERVE:—

7. The character of the lesions. Compare and contrast with those on the leaves; with those on the fruit.

8. The extent of the lesion on the cane; on the tendril. Are the lesions limited and definite? Is there a tendency to girdle?

The black rot lesions show sooner or later the characteristic **Signs** of the disease. Examine with the hand-lens or binocular the upper surface of the leaf spots and OBSERVE:—

9. The numerous minute black pimples protruding from beneath the epidermis. These are the pycnidia of the pathogene. What is the explanation

of their arrangement in the lesion? Do they occur on the under side of the lesion?

10. That these pycnidia also occur on the berry lesions and cankers. Compare as to numbers and type of arrangement with those on the leaf spots. In what part of the berry lesion do the pycnidia develop first. Why?

Make a series of DRAWINGS showing the symptoms and signs of the black rot on the different organs of the suscept.

The **Histologic Symptoms** of this disease are all necrotic in character. They may be satisfactorily studied only in fresh material. Thin free-hand sections through young lesions on leaf blade, petiole or berry are most serviceable. If material is available study and compare your findings with the observations of Reddick (1911 : 336–339); read carefully what he has to say on the matter. Make DRAWINGS to show your findings.

ETIOLOGY

The pyrenomycetous fungus, *Guignardia bidwellii* (Ellis) Viala and Ravaz, is the cause of the black rot of grapes. Its conidial form when occurring on the leaves has been given the genus name Phyllosticta, but when on the canes, Phoma. A variety of specific names have been applied to it. (See Reddick 1911 : 307.)

Life History. This pathogene exhibits in its life history all the typical spore forms and pathological phenomena of a large number of the leaf spotting and fruit rotting pyrenomycetes.

Primary Cycles are initiated in late spring and probably throughout the summer (Reddick 1911 : 310). The fungus winters largely in the mummies which lie on the ground or cling to the vines. Canes and tendrils may also harbor it.

Pathogenesis. Some of the mummies are provided for study. With the hand lens, OBSERVE:—

11. The very numerous pimple-like elevations,—the perithecia; the ostiolum at the apex. These are distinguishable from the pycnidia only by microscpic examination of their contents.

Under the hand lens or binocular remove with a needle one or more of these perithecia (from the soaked mummy provided) to a drop of water on a slide; cover and examine with the low power, exerting slight pressure on the cover-glass. NOTE:—

12. That if the perithecia are mature, the asci with ascospores will be forced out.

13. The average number of asci in the perithecium; the number of spores in an ascus.

DRAW an ascus with ascospores. These ascospores constitute in most seasons the chief inoculum for primary cycles. Discharged from the perithecia during rainy weather, they are blown by the breeze or splashed by rain drops to young leaves or berries where they germinate and the germtubes penetrate the suscept tissues. COPY Reddick 1911, pl. 5, fig. 28–29. Note the appressorium formed on the end of the germtube. Label in detail.

Pycnospores overwintered in mummies, canes and tendrils may also serve to initiate primary cycles. (See Reddick 1911 : 326.) With a needle, remove a few pycnidia from one of the cane lesions. Crush on a slide in water or KOH solution and study. MAKE OUT:—

14. The numerous pycnospores set free from the broken pycnidia. Their form, size and color as compared with the ascospores. DRAW. These germinate by a simple germtube and infect young leaves and fruits. COPY Reddick 1911, pl. 5, fig. 34. Label in detail.

After penetration by the germtube of the ascospore or pycnospore, mycelium is slowly developed in the surrounding tissue. The first evidences of infection appear in one to three weeks, depending on conditions of the suscept and the weather. After the production of the lesions, the pycnidia develop and form pycnospores with great rapidity. Make sections or study prepared slides provided and OBSERVE:—

15. Position of the pycnidium in relation to the tissues of the suscept; the intercellular mycelium.

16. The pseudoparenchymatous wall of the fruit body.

17. The short conidiophores bearing the pycnospores; their origin and arrangement in the pycnidium.

18. The ostiolum through which the pycnospores are extruded. They are discharged by the pressure resulting from water absorption by the gelatinous substance in which they are embedded. These initiate the secondary cycles.

Make a DRAWING to show the structure of the pycnidium and its relation to the tissues of the suscept.

Saprogenesis. The fungus lives saprophytically after the lesion is fully developed until the following spring. From July until October certain fruit bodies, known as spermagonia, are developed. They are abundant in August and may develop in the same stroma with pycnidia. They bear spores called spermatia. (See Reddick 1911, pl. 3, fig. 19.) Their function is unknown but it is believed by some that they are vestigial male elements. They have never been observed to germinate. Whether perithecia are produced in the old primary lesions is not certain. Pycnospores produced in these primary lesions may, as we have seen, winter over, and new ones may possibly be formed, thus completing the primary cycle. (See Reddick 1911 : 325–326.)

The **Secondary Cycles,** especially those initiated in the late summer differ from the primary cycles chiefly in that they give rise to the ascogenous structures which develop into perithecia the next spring.

Pathogenesis. Until the first of August, pycnospores are produced in enormous numbers on the mummied berries. They are discharged with rain or dew and initiate secondary cycles. Other cycles follow in succession as long as pycnospores are developed. After about August 1, pycnidial production ceases and instead there are developed in the lesions pycnidium-like bodies called pycnosclerotia.

Study slides provided or Reddick 1911, pl. 4. OBSERVE:—

19. The absence of a central cavity and of spores.

20. The parenchymatous character of not only the walls but also of the center of the fruit body.

21. Differences in the character of the central portion as contrasted with the wall structure.

READ Reddick 1911 : 329. DRAW to show the structure of the pycnosclerotium.

Saprogenesis. With the death and shriveling of the tissues in the berry, the fungus continues its activities as a saprophyte. The development of the pycnosclerotia (incipient perithecia) is halted by winter but resumed in late spring. In the thin walled parenchymatous-like center of the pycnosclerotium, asci are developed and matured. Study sections through mature perithecia or Reddick 1911, pl. 1 and pl. 5, fig. 26 and OBSERVE:—

22. The thick dark walls of the cells making up the wall of the perithecium; the mouth or ostiolum.

23. The rather thick walled asci; their shape, size and position in the perithecial cavity.

24. The ascospores; number and arrangement in the ascus.

DRAW to show the structure of the perithecium. The maturation and discharge of the ascospores completes the final secondary cycles.

References

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STRAWBERRY LEAF SPOT

There are at least three leaf spot diseases of strawberries in America but the one to which this name is applied is by far the most prevalent and injurious in eastern United States.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of this disease are for the most part necrotic in character. The hyperplastic development of anthocyanin in a narrow zone about the necrotic area is however one of the distinguishing symptoms of this leaf spot disease.

Leaf spot. The lesions on the leaves are the common and most numerous evidences of the disease. Examine the leaves provided and OBSERVE:—

1. The spots on the leaf, ranging from mere reddish flecks to definite circular areas with a necrotic center. Determine the average diameter of the dead areas.
2. The white center with its deep red or purple border. The width of the anthocyanescent zone.
3. That some of the spots coalesce to form lesions which may involve considerable areas of the leaflet.
4. The character of the under surface of the spots as compared with the upper surface. To what extent are the veins involved?

Petiole and pedicel cankers. Examine the material available and OBSERVE:—

5. The elongated spots on the petioles and on the fruit pedicels; the white centers of the lesions; which are very conspicuous. Are they surrounded by red margins as in the leaf?
6. The small and dried berries on the diseased pedicels. One or two berries may develop normally before the food and water supply is cut off, after which the others shrivel and are worthless.

The **Signs** of the disease are to be observed on the upper surfaces of the leaf spot. They are best observed in living leaves held for a few days in a moist chamber. Examine under hand lens or binocular, a spot on one of the leaves so treated and OBSERVE:—

7. The minute white tufts of slender conidia borne on short conidiophores.
8. The number and location of these tufts. Are they present in the anthocyanescent zone?

Make DRAWINGS to show the characters of the lesions on the different organs of the suscept.

Compare the morphologic symptoms of the leaf spot with those of the leaf blight (Anderson 1920 : 127–129, fig. 1) and the leaf scorch (Stone 1922 : 375–376, fig. 1) as shown by specimens and photographs. Make SKETCHES to contrast the symptoms of these diseases with those of the leaf spot.

The **Histologic Symptoms** of the disease may be studied in free-hand sections through lesions on the leaf or petiole. OBSERVE:—

9. The necrotic symptoms exhibited by the cells in the central area. Catalogue them.

10. The effect on the chloroplastids in the cells adjacent to the necrotic area.

11. The coloring matter (anthocyanin) in the tissues of the purple zone. In the cells of what tissues is the anthocyanin present. What other evidences of disease do you discover in the cells of this zone?

Make thin cross-sections through a canker on the petiole. DETERMINE:—

12. What tissues of the petiole are involved. Compare the morbid effects here with those observed in the leaf.

Make DRAWINGS of the sections to show the nature of the injury to cells in the diseased tissues compared with the conditions in the healthy tissues.

ETIOLOGY

The pathogene causing this disease is an ascomycetous fungus, *Mycosphærella fragariæ* (Tulasne) Lindau. Its conidial stage has long been known under the name *Ramularia tulasnei* Saccardo.

The **Life History** of this pathogene is in general typical of ascomycetous leaf spot fungi.

The **Primary Cycle** is initiated by conidia produced by the ascospores of the sexual stage found in the dead leaves on the ground.

Pathogenesis. The ascospores while still in the fruit body germinate, each sending out a long germtube which pushes forth through the mouth of the perithecium and produces a conidium at the tip. (See Dudley 1889 : 178–180.) Make a diagrammatic DRAWING to show production of primary conidia.

These conidia are blown to the young leaves where they each produce a germtube that directly penetrates the upper epidermis and invades the tissues beneath.

Cut out a diseased spot with the scalpel, and include a margin of the healthy tissue. Place the small piece in a casserole with a dropperful of potassium hydroxide solution, and boil over a micro-burner for a minute or so. Rinse two or three times with water, then mount and OBSERVE:—

13. The browned or blackened appearance of the diseased area in contrast to that of the healthy tissue immediately adjoining it.

14. The mycelium; inter- or intracellular? Its color, width, septation and branching. Does it extend beyond the necrotic area?

Make a DRAWING of the tissue as seen in this mount, showing the character of the mycelium and the extent of its penetration.

The hyphæ invade the tissues, using up the contents of the cells and permit the air to enter the tissues, thus giving the white appearance to the center of the lesion. These hyphal threads grow upward and produce a stroma ("sclerotium" of Trelease, Dudley 1889 : 175) just below the cuticle. The cuticle is then ruptured permitting the conidiophores to emerge (Dudley 1889, fig. 2 and 3).

With the aid of pith, cut thin sections through the diseased area of the leaf. Mount these in water (or use prepared slides) and OBSERVE:—

15. The location of the mycelium and its relation to the diseased cells.

16. The mat of mycelium or stroma just below the cuticle.

17. The three or more short stalks or conidiophores which arise from each stroma. Are they septate? A single conidium is borne on the tip of each conidiophore. The conidia will probably not be present in this mount. Why?

Make a DRAWING of a cross-section of a leaf showing the location and structure of the stromata with their conidiophores.

For study of the conidia, scrape several of the spots on the leaf with the scalpel and mount in water. OBSERVE:—

18. Their size, shape and septation. DRAW.

These secondary conidia give rise to the secondary cycles.

Saprogenesis. As the infected leaves die and droop upon the ground they are still farther invaded by the mycelium of the pathogene. This mycelium finally develops sexual fruit bodies or perithecia. These are at first covered by the tissue of the leaf, but soon break through the epidermis.

In the leaves provided OBSERVE:—

19. Small black bodies just large enough to be seen with the naked eye, near or in the old lesions. They are never numerous. Make a DRAWING (surface view) of part of a leaf showing these small back perithecia.

With the aid of pith, cut thin cross-sections of a leaf containing perithecia (or use prepared slides or see Dudley 1889, fig. 7). Under the microscope OBSERVE:—

20. That the perithecia are more or less hemispherical.

21. That the wall of the perithecium seems to be made of two layers, the outer one composed of thick-walled pseudoparenchymatous cells, and the inner one of thin-walled cells.

22. That there is an opening at the tip of each perithecium,—the ostiolum.

23. That arising from the inner base of the perithecium are club-shaped bodies,—the asci, containing eight hyaline, uniseptate spores with acute ends.

Make a DRAWING of a cross-section of the perithecium embedded in the surrounding tissue of the suscept. Make an enlarged DRAWING of a single ascus with its spores.

It is from these spores that the primary cycles start, the following spring. The mycelium may also live during the winter in the partially green leaves and produce conidia the following spring, thus initiating primary cycles. This is probably quite as often the means of wintering over as that afforded by the perithecial stage.

The **Secondary Cycles** are originated by the conidia from the diseased leaves which are splashed by rain drops to nearby healthy leaves, petioles, and pedicels.

Scrape conidia from lesions on the living leaves kept for some time in the moist chamber. Germinate in water on a slide and study germination. OBSERVE:—

24. The point of origin of the germtube; number from each spore and from each cell. Draw.

As in the case of the primary conidia from the ascospores these secondary conidia send their germtubes directly through the upper epidermis into the tissues beneath.

The pathogene completes its development in the secondary cycles by the production of perithecia if the infected leaf is killed or it hibernates as mycelium or stromata in the living leaf and produces conidia the following spring.

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CERCOSPORA LEAF SPOT OF BEETS

ALTHOUGH this disease is found more often on sugar beets, it also affects the common garden beet and Swiss chard. The disease is typical of a great many Cercospora leaf spots on different plants.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of this disease are typical of leaf spot diseases in general. They are chiefly necrotic The lesions are usually confined to the leaves but have been observed on the petioles as well as on the bracts and peduncles of the flower and on the seed balls.

Leaf spot. In the material provided OBSERVE:—

1. The spots of various sizes. Determine their average diameter. Do they ever coalesce?

2. Their shape and distribution over the leaf. Where most numerous?

3. The brown or greyish necrotic centers surrounded by a red or purple zone (often brown in dried specimens). This anthocyanescent zone is a common accompaniment of necrotic lesions in leaves.

4. The differences in the character of the upper and lower surfaces of the lesion.

5. That in some of them the necrotic center has cracked and parts have fallen out giving a ragged shot-hole effect.

As the diseased leaves drop off, new ones are formed, thus producing an elongated crown on the root. (See photographs or Duggar, Fungous Diseases of plants, p. 310.)

Make DRAWINGS to show the symptoms observed.

The **Signs** of the disease are to be observed under the hand-lens or binocular on the necrotic center of the spot. They are best seen on fresh leaves kept for a time in moist chamber. Examine such material as is available and MAKE OUT:—

6. The numerous minute olivaceous tufts scattered over the surface of the lesion. These consist of the fasciculate conidiophores bearing the long olivaceous conidia.

Make a DRAWING to show the appearance of these tufts as seen under low magnification.

The **Histologic Symptoms** of the disease may only be studied satisfactorily in fresh material. Make a number of thin free-hand sections through a lesion so as to include the anthocyanescent border and some of the adjacent healthy tissues. OBSERVE:—

7. The necrotic symptoms exhibited by the cells in the dead tissues. Catalogue them.

8. The effect on the chloroplastids in the cells adjacent to the necrotic area.

9. The coloring matter (anthocyanin) in the tissues of the purple zone. In the cells of what tissues is the anthocyanin present? What other evidences of disease do you discover in the cells of this zone?

Make a DRAWING of parts of the section to show these symptoms.

ETIOLOGY

The pathogene responsible for this disease is *Cercospora beticola* Saccardo, one of the hyphomycetous fungi. Only the asexual form of the fungus is known.

The **Life History** of this pathogene is a simple one as only one spore form is involved and secondary cycles are essentially repetitions of the primary.

The **Primary Cycles** are initiated by conidia developed on overwintered tops and crowns left in the field. They are blown by the wind or spattered by the rain to the first young leaves of the season's planting.

Pathogenesis. While the conidial inoculum for the primary cycles is produced on the overwintered débris of the suscept, (see Pool and McKay 1916 (b), fig. 1) the conidia which function in this rôle are not different from those more readily obtained from lesions on living leaves. We will use the latter instead.

Scrape several of the spots with a wet scalpel and make a mount of the material thus procured in water. Under the microscope OBSERVE:—

10. The thin, long, slender spores, which are slightly thickened at one end and tapering at the other. How many septa in each? DRAW.

These conidia germinate and the germtubes enter the leaf through the stomata.

If fresh material is available germinate the spores (if not see Pool and McKay 1916 (a), fig. 6). OBSERVE:—

11. The number of germtubes developed from each spore; number from each cell. DRAW. Study leaf invasion through the stomata as shown by Pool and McKay 1916 (a), pl. 81. Copy one or more of the figures, A, B, C, or F. Label in detail.

The mycelium developed from these germtubes spreads through the tissue and kills it. A leaf lesion or spot thus results. Very shortly this mycelium develops the fruiting structures on the surface of the lesion.

Cut out a spot from the leaf including a margin of healthy tissue. Boil in a casserole for half a minute in KOH solution over a microburner. Mount in water and OBSERVE:—

12. At the edges of the lesion, the almost hyaline branching mycelium, which may be uniform in width or have occasional cells thickened, with granular vacuolated contents. DRAW.

13. The tufts of brown conidiophores closely crowded at the base and widely separated at the top.

14. That very few of the conidiophores are straight but appear knotted or angled.

15. That at each angle is a scar where a conidium had been borne. How many scars does each conidiophore have?

16. The sclerotia-like mass of cells from which the conidiophores arise; color, size. Its relation to the stoma.

Place a lesion, lower surface up, under the binocular microscope and OBSERVE:—

17. The points emphasized in 13 and 14.
18. The relation of the tufts of conidiophores to the stomata.
19. The attachment of the conidium to the conidiophore. Is the thickened or the thin end attached to the stalk?

Make a DRAWING showing the points brought out under paragraphs 12 to 19.

Saprogenesis. No perfect stage of this fungus has yet been found. The fungus continues to fruit for a time after the leaf tissues die. The sclerotia-like bodies from which the conidiophores arise remain alive under favorable conditions, in the débris of the suscept. They may produce a new crop of conidia the next spring which may give rise to the primary infections.

Secondary Cycles are initiated during the summer by conidia from primary and secondary lesions. They repeat in detail the phenomena of the primary cycle.

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SEPTORIA LEAF SPOT OF CELERY

THIS disease is often called late blight in contradistinction to the early blight or Cercospora leaf spot. Both of them will affect the plant while it is still in the seedling stage but the Septoria leaf spot is usually most severe late in the season. Both diseases are very destructive to the celery crop.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of the disease are all necrotic. Lesions occur on all the above ground parts but are most common on the leaves and petioles. When celery is allowed to go to seed the inflorescence may also show lesions.

Leaf spot. Examine specimens provided and OBSERVE:—

1. The numerous spots scattered over the leaf, both on the upper and lower surface. Do the lesions extend through the leaf? Relation to leaf veination.

2. That the spots are irregular in outline and often coalesce with neighboring spots. Do they have any definite zones surrounding them as found in lesions on strawberry or beet leaves?

3. The color of the necrotic area.

4. The average size of the spot. Plot a curve to show the frequency of variations in the diameter of 50 or more spots.

The **Signs** of the disease are the black papilla-like pycnidia developed in the necrotic centers of the spot. Examine the spots under a hand lens or the binocular. OBSERVE:—

5. On which side of the spot these pycnidia are developed. Are they superficial or embedded in the tissues?

6. Number per spot. Calculate the average from countings on 10 or more spots.

The presence of these pycnidia serve to distinguish this disease from the bacterial leaf spot. Compare lesions of the two diseases.

Make a DRAWING to show the signs and symptoms of this disease on the leaves.

Petiole spotting. In the material provided OBSERVE:—

7. The sunken brown areas on the petioles. How do these compare with the lesions on the leaves as to size, color and form?

8. The pycnidia, their relative abundance and distribution as compared with those on the leaf lesions.

DRAW to show the signs and symptoms on the petioles.

Seed spot. If specimens of diseased seed capsules are not provided, see Klebahn 1910, fig. 1 or Coons and Levin 1916, fig. 4. COPY.

The **Histologic Symptoms** may be profitably studied only in fresh material. Make a number of freehand sections through a lesion on the leaf or petiole. OBSERVE:—

9. The necrotic symptoms exhibited by the cells in the necrotic area. Catalogue them.

10. Evidences of injury to the cells in the living tissues adjacent to the dead tissues. What are they?

Make a DRAWING to show the histologic changes exhibited by this disease.

ETIOLOGY

The pathogene is *Septoria petroselina* Desmazieres var. *apii* Briosa and Cavara. It is one of the so-called imperfect fungi.

Life History. Only the conidial form of the pathogene is known. In this form it is able to survive the winter, hence a sexual stage, if it does exist, is probably of little importance in the perpetuation of the fungus.

The **Primary Cycle** has its beginning with infection by the conidia which are present in the débris of last year's crop, or with those which are found on the seed.

Pathogenesis. Crush in water several of the small black pycnidia from old leaves, cover and OBSERVE:—

11. The thin long cylindrical spores, slightly curved and tri-septate. (Staining with iodine may be necessary to make the septation evident.)

If fresh material is available cut out a bit of the lesion showing pycnidia. Chop and crush in a drop of water on a slide to free some of the spores. Allow them to germinate. OBSERVE:—

12. The manner of germination; number and origin of the germtubes.

Make a DRAWING of a germinating spore.

These spores are blown about by the wind or spattered by rain to the outer, lowest leaves of the celery, where they germinate, sending germtubes through either the upper or lower epidermis. The mycelium which is usually intercellular, soon forms pycnidia.

Cut thin cross-sections of a diseased spot or use prepared slides and under the high power, OBSERVE:—

13. The large thick-walled pycnidium, which ordinarily is nearly globose. Does it set deeply into the tissues?

14. The pseudoparenchymatous nature of the pycnidial wall, and the round opening,—the ostiolum.

15. Lining the pycnidial cavity, the short, sharply pointed conidiophores on the ends of which the slender conidia are borne.

16. The mycelial threads penetrating the tissue all about the pycnidium; the septation, contents and the branching of this mycelium.

Make an enlarged DRAWING of a pycnidium with the surrounding suscept tissues.

Saprogenesis. Klebahn has shown that there may be vegetative activity during the saprogenic phase. The fungus usually winters over as spores in the pycnidia in the old leaves and stems which have been left in the field. These spores serve as the inoculum the following spring.

Secondary Cycles. The conidia from the diseased leaves ooze from the pycnidium whenever moisture is present and are transferred by rain, tools or animals to the stems or other leaves. Secondary infections are repeated until the suscept is killed or harvested.

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BLACK SPOT OF ROSES

THE black spot is a disease peculiar to the rose, and appears wherever this ornamental is grown, in temperate and tropical regions, in greenhouse and garden. It is probably the most serious disease of roses. Few varieties appear to be immune.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of the black spot are chiefly of the plesionecrotic type. The lesions are largely confined to the leaves though they may appear on the current year's growth of the shoots (Alcock 1918).

Leaf blotch. Study the material provided and OBSERVE:—

1. That some of the affected leaflets show isolated, more or less circular black areas.

2. That they do not at first extend through the leaf.

3. That the margins of these spots are radiate fibrillose. Study with the hand lens.

4. The distribution of the lesions. Do they occur on both sides of the leaflet? Are they marginal or located more toward the center of the blade?

5. The indefinitely delimited yellow zone surrounding many of the blotches, evidence of the disorganization of the chlorophyl in the tissues beyond the advancing margin of the pathogene.

6. That some leaflets are nearly or quite covered by the lesions. Explain how this comes about.

Make a DRAWING of one or more leaflets to show these symptoms.

7. That the lesions vary somewhat on different varieties of roses. In what respects? Show this in SKETCHES. Label to bring out these differences.

The leaves of some varieties become yellow very soon after the spots develop. In others there is little or no yellowing but in either case premature defoliation occurs. The early loss of foliage weakens the plants.

The **Signs** of this disease while evident to the naked eye are more easily recognized under the hand lens or binocular. Examine the surface of an old lesion and OBSERVE:—

8. The minute flat circular fruit bodies—the acervuli scattered over the surface.

9. The radiating strands of subcuticular mycelium or rhizomorphs upon which the acervuli are formed.

10. The cracked cuticular covering over the fruit bodies.

Make an enlarged DRAWING to show the surface view of the acervuli and subcuticular mycelium.

Shoot blotch. If material is available study the symptoms of the disease as exhibited by the shoots. If not, see Alcock 1918, pl. 6. DRAW to show the characters of the stem lesions.

The **Histologic Symptoms** of this disease can be advantageously studied only in fresh material. Make thin sections through the margin of a young lesion on the leaf. DETERMINE:—

11. What morbid changes have occurred in the epidermal cells. (Compare with adjacent healthy cells.) Catalogue them.

12. What morbid changes have been produced in the palisade and mesophyl cells.

Make thin sections through the yellow zone including some of the adjacent green tissues. DETERMINE:—

13. What changes have occurred in the chloroplasts in the yellow zone.

14. What morbid changes if any are to be observed in other organs of the cell.

Make DRAWINGS to show the histologic symptoms.

ETIOLOGY

The cause of this disease is an ascomycetous pathogene, *Diplocarpon rosæ* (Libert) Wolf, the conidial form of which has long been known under the name of *Actinonema rosæ* (Libert) Fries.

Life History. The life activities of this pathogene are perfectly correlated with the seasonal changes of the temperate zone although the family, Microthyriaceæ, to which this fungus belongs, is largely tropical.

The **Primary Cycles** develop on the first leaves put forth by the bushes in the spring. In tropical or semi-tropical countries where roses have no resting period or in the greenhouse, only secondary cycles occur.

Pathogenesis. In northern regions the inoculum for primary cycles appears to consist chiefly of ascospores developed in fruit bodies in the overwintered leaves on the ground. That conidia may also function as primary inoculum has also been shown to be probable by Alcock (1918) who found them in the spring in abundance on shoot lesions and on living leaves that had remained on the bushes over winter. We shall here confine our studies to the ascospores as inoculum for the primary cycles.

Remove a bit of tissue containing perithecia from the overwintered leaves (or if not available study the drawings by Wolf 1913, pl. 1, fig. 6–7). Chop and crush the leaf tissue so as to free the ascospores. OBSERVE:—

15. The long sack-shaped asci each containing a number of ascospores. How many?

16. Some of the free ascospores in the water here and there; shape, color and number of cells in each.

Make DRAWINGS of some of the ascospores.

The ascospores do not appear to be shot into the air as is usually the case but simply ooze out on the surface of the old leaf. How may they reach the unfolding leaves of the plant? These ascospores will germinate only on the surface of living rose leaves.

Study ascospore germination as shown by Wolf (1913, pl. 1, fig. 9). OBSERVE:—

17. The number of germtubes from each spore. DRAW.

The germtube penetrates the cuticle and gives rise to hyphæ which, spreading just beneath it over the epidermal cells, form the radiating strands of subcuticular mycelium.

To study the structure and arrangement of these subcuticular strands, use prepared stained surface mounts. OBSERVE:—

18. The flat radiating mycelial strands or rhizomorphs. How many hyphæ in a strand?

19. Septation of the hyphæ and manner of branching of the rhizomorphs.

DRAW to show characters of this subcuticular mycelium.

Make thin cross-sections through a spot showing numerous acervuli. Stain with methylene blue, wash, cover and OBSERVE:—

20. The relation of the acervuli to the leaf tissues; subepidermal, subcuticular, or on the surface? (Compare with prepared stained slides.)

21. Structure of acervulus; conidiophores and conidia.

22. Mycelium, in epidermal cells and palisade tissues, beneath the acervulus; inter- or intracellular?

Make a detailed DRAWING of a section through the acervulus.

These conidia, splashed by rain to healthy leaves, germinate to initiate the secondary cycles.

If fresh material is available study and DRAW; if not COPY Wolf 1913, pl. 1, fig. 2–3.

Saprogenesis. After the diseased leaves fall to the ground, the subcuticular mycelium forms peculiar shield-like structures, (see Wolf 1913, pl. 1, fig. 4) beneath which in the leaf tissue below the epidermal cells, the mycelium forms a perithecium. These perithecia begin to form in the autumn but do not mature until spring.

If prepared sections through perithecia in the leaf are available (if not see Wolf 1913, pl. 1, fig. 6 and 7) study and OBSERVE:—

23. The pseudoparenchymatous walls of the perithecia. Of what does this consist?

24. The hymenium within, made up of asci and paraphyses.

25. The two-celled ascospores in the ascus. DRAW a single ascus with accompanying paraphyses.

26. The cuticle and epidermal cells of the leaf.

Make a SKETCH showing the structure of a perithecium in section.

Secondary Cycles. These are initiated repeatedly throughout the season by conidia from acervuli. Leaves involved in the secondary cycles fall prematurely like those suffering from the primary infections and the next spring also produce a crop of ascospores.

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BITTER ROT OF APPLES

Although bitter rot is known to occur on many different plants it is of most importance on the apple in the southern and central states. It is practically unknown in northeastern United States. It has been estimated that American growers of this fruit lose from bitter rot in some years $10,000,000. For the most part the disease has been called bitter rot, but the names, anthracnose and ripe rot, have also been used.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of the bitter rot are primarily necrotic. Lesions occur so far as known only on the fruits and bark of the tree.

Cankers. The lesions in the bark are typical cankers. They occur commonly on limbs, twigs and even fruit spurs but rarely on the body of the tree. In the specimens provided OBSERVE:—

1. The sunken canker area; its sooty black color; the delimiting crack more or less continuous about the lesion.

2. The checked and cracked bark within the cankered area. (Von Schrenck and Spaulding 1903 : 31–32 and Roberts 1918, pl. 3.) Compare in this respect with a fire blight canker.

3. That there is commonly the remains of a blighted twig or spur near the center of the canker (Von Schrenk and Spaulding 1903, pl. 7–8).

4. The swollen appearance of the limb at the point where old cankers occur. This is due to callus formation in an attempt of the tree to heal out the canker (Burrill 1907, pl. 1, fig. 2).

When the twigs are girdled and killed a twig blight symptom develops. Twig blight occurs to a marked extent only on certain varieties like the Jonathan and Willow.

The characteristic **Signs** of the disease are usually to be discovered on the cankers. Examine the specimens carefully and OBSERVE:—

5. Numerous small black pustules breaking forth through the epidermis in the cankered area. These are the fruit bodies,—acervuli, of the pathogene. They are most prominent in young stages of the canker (See Von Schrenk and Spaulding 1903, pl. 9, fig. 4–6).

DRAW to show the canker symptoms and signs.

Fruit rot. Spores washed from the cankers by rain spatter on to the growing fruit causing the long known bitter rot of the apple. Fruit rot ordinarily appears in July and August, although it may be seen as early as June and as late as October. Lesions appear as light-brown discolorations beneath the skin. They are at first very small but soon enlarge and become firm in texture and circular in outline. When the lesions are 3 mm. or more in diameter, the surface is distinctly sunken. Usually each fruit shows only a few spots, but in cases of severe infection more than a 1000 separate lesions may occur (Roberts 1915, pl. 7, fig. 1). In cases like the latter few if any of the spots increase in size, but instead, after reaching a diameter of about 2 mm., cease enlarging and appear as brown raised blisters. Study the specimen provided and OBSERVE:—

6. The stage of development of the affected apple. At what stage, green or ripe, was the fruit affected?

7. The spots on the fruit; their size, color, distribution, appearance (sunken or raised). Estimate the number on the apple.

In more typical cases of bitter rot only a few spots occur on each fruit (see Von Schrenk and Spaulding 1903, pl. 2). These spread rapidly, involving the entire fruit in a short time. Ripe apples have been inoculated with pure cultures of the fungus and placed in an incubator at 30° C. Study the inoculated apples and OBSERVE:—

8. The size of the lesion; its form, and color. Note any other characters exhibited.

The **Signs** of the disease are usually striking on the large rot lesions on the fruit. OBSERVE:—

9. The concentric rows of minute pustules,—the acervuli, bursting through the epidermis. The oozing spore mass; its color.

Make DRAWINGS to show the two types of fruit lesions and an enlarged DRAWING of one of the rot lesions to show the acervuli (see Von Schrenk and Spaulding 1903, pl. 6, fig. 2).

Cut the diseased apple longitudinally through one of the rotten areas. OBSERVE:—

10. The depth of the rot, color of the infected tissues, and taste. Is it bitter?

The entire fruit finally becomes involved and gradually shrivels to form a mummy which falls or remains clinging to the tree. Examine the mummied fruits provided and make a SKETCH of one.

Make a DRAWING to show the internal symptoms of the fruit rot.

To study the **Histologic Symptoms** make thin free hand sections through a piece of the diseased fruit cut so as to include both rotted and healthy tissue. Compare the conditions in the diseased tissues with those in the healthy. LIST the histologic changes to be observed.

ETIOLOGY

The pathogene, now known as *Glomerella cingulata* (Stoneman) Von Schrenk and Spaulding, is an ascomycetous fungus belonging to the family Gnomoniaceæ of the Pyrenomycetes. Its conidial stage has long been known under several names, that usually applied to the fungus on the fruit being *Glœosporium fructigenum* Berkley.

Life History. Extensive studies on the life history of this fungus have been made, especially by American workers, so that the details are now well known.

Primary Cycle. As the pathogene requires a high temperature for development the primary infections appear late, seldom before July.

Pathogenesis. The mummied fruits hanging on the trees or lying on the ground and the cankers are the sources of the primary inoculum. The fungus hibernates in these, producing conidia during the spring in acervuli. Apparently the ascospore stage of the fungus which occasionally develops in both the mummies and cankers plays but a small rôle in the life history of this pathogene. Make crushed mounts of acervuli from the mummies or cankers provided and OBSERVE:—

11. The hyaline conidia; their form, granular contents, abundance and variation in size. DRAW several to show these points.

These conidia washed and splashed by the rains to young fruits, initiate primary infections. Germinate the conidia. OBSERVE:—

12. The long septate germtubes; point of origin.

13. The dark thick-walled appressoria formed at the tips of some of the germtubes. What is their function? (Compare Von Schrenk and Spaulding 1903, pl. 5.) DRAW several germinating spores.

The germtubes, directly from the spores or from the appressoria, penetrate the uninjured skin of the apple or gain an entrance through wounds and a mycelium is rapidly developed resulting, usually within a few days, in a distinct rot lesion. General infection of the fruits is often in a conical region beneath a mummy or canker. Remove a bit of the diseased apple flesh with the forceps and STUDY:—

14. The mycelium; its character and branching. DRAW.

The lesions soon begin to show developing fruit bodies,—the acervuli of the conidial stage of the fungus.

Study the diseased fruits provided. NOTE:—

15. The location and concentric arrangement of the acervuli within the lesion.

16. The different degrees of development of these fruit bodies from the center toward the margin of the spot.

17. The mass of conidia oozing from each mature acervulus; color and consistency of the mass. Remove a spore mass to a drop of water; note the dissolving effect. When dried the conidial mass is hard and horny. The conidia are held together by a gelatinous substance which quickly dissolves in water.

So long as the conidia remain dry they retain their viability. Once wetted and scattered they either germinate or if promptly dried again they lose their viability.

Make a number of very thin sections through a bit of the affected tissue showing mature acervuli (or use prepared sections). Study and OBSERVE:—

18. The great mass of conidia pushed forth through the ruptured epidermis, the recurved edges of which show at each side of the spore mass.

19. The mycelial bed, forming the base of the acervulus, from which the short simple conidiophores arise. How are the conidia formed?

20. The mycelium spreading throughout the adjoining tissues; inter- or intra-cellular?

DRAW to show the structure of the acervulus and its relation to the tissues of the suscept.

Saprogenesis. With the mummification of the fruit and the death of the bark in the cankers, the mycelium does not die but sometimes continues a saprophytic existence. It lies dormant through the winter, resumes its activities in the spring and usually produces acervuli, though sometimes the sexual fruit bodies, perithecia are developed. The latter are rare. Acervuli having been studied, a brief examination of the perithecium may be made. Study the prepared sections. OBSERVE:—

21. The long blunt protruding beak of the perithecium.
22. The relation of the perithecium to the tissue of the suscept.
23. The asci; form and arrangement in the perithecium.
24. The structure of the perithecial walls.
25. The ascospores; number and arrangement in as ascus; form.

DRAW to show a longi-section of the perithecium. The ascospores, when produced, doubtless serve to initiate primary cycles.

Secondary Cycles are initiated repeatedly throughout the growing season on fruits and twigs, causing fruit rot, twig blight and limb cankers. The pathogene exhibits the same structures and phenomena as in the primary cycles.

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APPLE BLOTCH

THIS disease is generally known as apple blotch, but it is still sometimes called black scab, fruit blotch, and star fungus. It should be clearly borne in mind that apple blotch is entirely distinct from sooty blotch and apple scab, in spite of the common names applied. This disease, like bitter rot, is confined largely to central and southern United States. In the southern half of the apple belt it is more destructive, taken year by year, than bitter rot and apple scab combined.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of the apple blotch are typically necrotic, the dominant lesions being cankers, leaf spot, and fruit blotch. There is no rotting of the fruit tissues as in black rot or bitter rot.

Cankers. The disease affects the fruit spurs, twigs and rapidly growing shoots, producing characteristic cankers. Larger limbs and trunks are not usually affected. In the material provided, OBSERVE:—

1. That on the fruit spurs, the spots are at first purplish or blackish. The center becomes tan colored, then grey with age, the margin remaining purplish. The resulting lesion is small but rather conspicuous, with a crack along the margin.

2. That on water sprouts and on other rapidly growing shoots, the cankers have much the same appearance as that just described, but are larger, sometimes measuring an inch or more in length and girdling the stem. Longitudinal cracks appear not only along the edge but also through the cankered tissues, eventually giving the surface a rough scurfy appearance. This character is noticeable on cankers two or three years old. The canker may extend at several points along the margin, producing a lobed or somewhat concentric appearance. Frequently the canker is limited permanently by a crack and the wound heals out (see Gardner et al. 1923 : 7–8, fig. 5, 6, 10; Scott and Rorer 1909 : 10–11, pl. 1).

Make DRAWINGS to show characters of the cankers on twigs and spurs.

3. The somewhat swollen condition of the twig under old canker lesions. This is a hyperplastic symptom due to the callus formations beneath the dead bark.

4. The depth to which the canker extends. Does it reach the cambium?

5. Relation of the twig cankers to leaf scars (see Gardner et al. 1923 : 11–12).

Leaf spot. Examine the leaves provided (also Scott and Rorer 1909, pl. 1 and Gardner et al. 1923, fig. 7). OBSERVE:—

6. The remarkably small size of the spots; their numbers and distribution over the leaf blade.

7. Shape and color of the spots; appearance on both sides of the blade.

8. The lesions on petioles and mid ribs. How do they differ from those in the blade.

Defoliation may result from severe leaf spotting but usually the injury is insufficient to cause fall of the leaves. Petiole lesions may cause the leaves to fall prematurely or to turn brown on the tree.

DRAW to show typical leaf lesions.

Fruit blotch. The fruit lesions are typical blotches. They vary somewhat in appearance in different varieties. Examine the diseased apples available as well as the illustrations in the bulletins cited. OBSERVE:—

9. That the first evidence of the disease is a very small inconspicuous, light-brown speck, which, under slight magnification has the appearance of a stellate collection of brown fibers just beneath the epidermis. On account of this stellate character of the spot this disease is at times confused with apple scab. On young apples, the lesions are at first water-soaked, and in wet weather there may be a yellowish gummy exudate therefrom.

10. That spots enlarge radially, attaining a diameter of from 3–10 or more millimeters, becoming darker in color. On light-colored varieties the spots are green, later becoming brown, while on red varieties the spots are at first red, then changing to black.

11. The irregular lobed and fimbriate margins of the lesions.

12. That sometimes the spots are sunken and may or may not show this fringed margin, or they may be raised; gradations between the fringed smooth spot and the umbonate spot (raised and strongly projecting in the center). Where the spots are numerous, they may coalesce, forming large lesions involving a major portion of the fruit.

13. The cracking of the surface of badly diseased fruits. To what is this due?

14. The depth to which the lesion extends.

Make DRAWINGS to show the fruit lesions.

The **Signs** of this disease are rather inconspicuous but taken together with the symptoms they are diagnostic for the blotch. Examine again (best seen under a hand lens) the surface of the cankers, leaf spots and fruit blotches. OBSERVE:—

15. The minute black pimples, the pycnidia of the pathogene.

16. Their relative abundance in the lesions on twigs, leaves and fruits.

INDICATE the presence and distribution of the pycnidia in the drawings already made.

ETIOLOGY

The pathogene causing apple blotch is known as *Phyllosticta solitaria* Ellis and Everhart. This name applies to the asexual stage, no sexual stage being known. It may be regarded as an ascomycete, even in the absence of a perithecial stage, but until the missing stage is found, it is temporarily grouped with the Fungi Imperfecti (Guba 1924 : 234).

Life History. A number of important links in the life history of this pathogene apparently remain to be discovered. Enough, however, is known to account for its whereabouts throughout the year.

The **Primary Cycles** are initiated rather late in the spring, usually a month or more after the petals fall.

Pathogenesis. Twig cankers are the chief sources of the inoculum. The fungus hibernates in these lesions as mycelium which becomes active in the spring and which produces pycnidia along the advancing margin of the canker. Examine the twigs provided. OBSERVE:—

17. The minute black pycnidia in the bark near the margin of the cankers. DRAW to show their appearance under a hand lens.

Crush a bit of the bark containing pycnidia and examine for pycnospores. OBSERVE:—

18. That they are ovoid elliptical, hyaline, one-celled. DRAW.

19. That they sometimes exhibit a gelatinous sheath, as shown by Scott and Rorer (1909, pl. 3, fig. 2–5). COPY to show this. These pycnospores are washed or carried to the young fruits, twigs and leaves.

If fresh material is available set conidia to germinate. Germination of these pycnospores occurs in twelve to eighteen hours. Examine the germinating spores and OBSERVE:—

20. Number and position of the germtubes; branching, septation and contents. DRAW.

The mycelium developed from the germtubes spreads through the superficial tissues of the fruit in a stellate fashion as may be observed by a careful examination of the lesions. Pycnidia are soon developed by this mycelium beneath the epidermis. Study prepared slides showing the pycnidia in section. OBSERVE:—

21. The position and relation of the mature pycnidium to the tissues of the suscept.

22. The structure of pycnidial walls and conidia-bearing structures.

Make a detailed DRAWING of the pycnidium and adjacent suscept cells.

The pathogene produces only sterile pycnidia on the leaves. Apparently on these organs the cycle is never completed. The pycnospores, matured in primary lesions on twigs and fruits, serve to initiate secondary cycles. In the twig lesions, the pathogene hibernates either as immature pycnidia (pycnosclerotia) or as mycelium from which pycnidia are developed in the spring. Thus is the cycle completed without the appearance of a sexual spore form.

Saprogenesis. There is no marked tendency exhibited by this pathogene to grow and develop saprophytically. It may feed to some extent on the dead tissues after leaves and fruit have fallen. The formation of spore producing pycnidia only during the fore part of the season, followed by the development (in the later secondary cycles at least) of pycnosclerotia, suggests that perithecia, similar to those known for *Guignardia bidwellii* on grapes, may be produced during saprogenesis. They are as yet unrecorded (Guba 1924 : 235).

Secondary Cycles originate from the pycnospore crop of the primary infections. They are initiated only during the first half of the summer, and probably do not often initiate other secondary cycles. They duplicate in all respects the primary cycles.

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BLACK ROT OF APPLES

WHILE this disease affects chiefly the apple, other pomaceous fruits are by no means free from the trouble. Marked variation in susceptibility of varieties is exhibited. In western New York the Twenty Ounce is known as the variety most susceptibe to the canker form of the disease. As a disease of the fruit and foliage it is of little importance in the north but is a major disease of apples in the southern apple growing sections.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of this disease are necrotic in character. Lesions appear on limbs, leaves and fruits, known on each respectively as canker, leaf spot and fruit rot.

Cankers. The larger limbs are more often affected than the twigs and trunk, though body cankers frequently develop on the trunk following so-called winter injury. The first evidence of canker formation on large limbs is a reddish brown sunken area in the bark. (See Hesler 1916 : 62–63 and pl. 8.) Study the cankered limbs provided and OBSERVE:—

1. The shape, size, color and margins of the canker.

2. The surface; rough, checked and cracked. Compare with fire blight cankers.

Make a DRAWING of the canker to show these symptoms.

Leaf spot. The first evidence of the disease on the leaf is the appearance of minute purplish specks which soon enlarge until they reach a diameter of 2 to 10 mm. the centers of which soon become necrotic. Examine the specimens provided and OBSERVE:—

3. The form of the spots. At first circular, they often become lobed later. To what is this due? The term "frog-eye" is frequently applied to such enlarged leaf lesions. This type of spot is most commonly met with in the southern range of the disease (see Crabill 1915, fig. 4 and cover picture).

4. The color of the necrotic area. Note that the margin is very sharp and definite.

5. The size of the spots. Determine the average diameter of 20 lesions.

DRAW to show the character of leaf lesions. Defoliation often follows severe attack (Scott and Rorer 1908, pl. 4).

Fruit rot. Black rot is primarily a rot of ripe fruits, though green apples may show the lesions. Study the material provided (also Hesler 1916, pl. 7, fig. 1). OBSERVE:—

6. That the lesions appear first as small brown spots which may turn black very soon or remain brown until the entire fruit is involved. Why this difference?

7. The concentric color zones in the lesion.

8. The firmness of the rotted tissues; color, taste and odor.

9. That the lesion usually starts at some wound, as the hole made by the codling moth. Of what significance is this?

10. That the lesion is sometimes formed at the blossom end; a type of injury common in the south. (Crabill 1915, fig. 5 and Hesler 1916, pl. 12, fig. 2.)

11. That finally the fruit becomes entirely involved and slowly shrivels to form a hard black mummy (Hesler 1916, pl. 7, fig. 2) which may hang to the tree over winter.

Make DRAWINGS to show the various stages of fruit rot.

The **Signs** of the disease are the minute black pimple-like pycnidia. They are formed in great abundance especially on the cankers and fruit lesions. They are less often to be discovered in the leaf lesions. Re-examine the cankers and fruit lesions under a hand lens or binocular. OBSERVE:—

12. The numbers and distribution of the pycnidia. Show this point in the DRAWINGS of cankers and fruit lesions which you have made.

The **Histologic Symptoms** may be studied to advantage only in fresh material. Cut thin section through the margin of a lesion in the apple. Under the microscope OBSERVE:—

13. The changes which have taken place in the cells in the diseased portion in comparison with those in the healthy tissues. Catalogue these in terms of histologic symptoms exhibited. Show these in a DRAWING of the section.

Histologic changes in leaf and bark lesions can best be understood by a study of Hesler's work on these lesions (1916 : 109–116). Make SKETCHES to illustrate the facts set forth. Label fully.

ETIOLOGY

The name of the pathogene is *Physalospora cydoniæ* Arnaud. It is an ascomycete but was long known only in its pycnidial stage as *Sphæropsis malorum* Berkley, under which name it is still frequently referred to in literature. It is closely related to *Guignardia bidwellii*, the pathogene of black rot of grapes.

Life History. This pathogene as indicated by its habits is evidently a facultative parasite. It grows most vigorously as a saprophyte on dead plant tissues. It depends, almost entirely on its asexual spores for propagation at least in the north. The perithecial stage seems to be rare and has been found chiefly in specimens from relatively warm regions.

The **Primary Cycles** appear to be initiated chiefly by pycnospores. Since ascospores seem to be so rarely produced, they may be largely disregarded as inoculum.

Pathogenesis. The source of pycnospore inoculum is the dead tissues of the suscept in which the pycnidia formed the previous season have over-wintered. The dead bark of cankers is a common source. Old mummies which have clung to the tree may also serve to harbor the pathogene. Twigs killed by fire blight are promptly invaded by this fungus and pycnidia gorged with spores are formed during the latter part of the season. Such twigs are frequent sources of inoculum. The overwintering mycelium in active cankers may also form pycnidia full of spores in the spring.

Remove several of the pycnidia from one of the sources provided. Crush in a drop of water on a slide. Examine under the microscope and OBSERVE:—

14. The great number of pycnospores oozing from the crushed pycnidia. Estimate the number produced by a single pycnidium.

15. The size, color, shape and septation of the pycnospores. DRAW to show variations in spore form, coloring and septation. They become two celled only with age.

Splashed by rain or possibly carried by insects to leaves or wounds in fruit or bark these pycnospores quickly germinate and initiate infection.

Germinate some of the spores in water and OBSERVE:—

16. The long germtubes which are soon put forth. How long did it take the spores to germinate?

17. The color, contents, septation and branching of the germtubes. How many from each spore? DRAW.

From the infection court the germtube penetrates the suscept and rapidly develops into a branching mycelium, which spreads in all directions through the tissues. To study this mycelium in its relations to the suscept cells, mount a bit of rotted fruit well teased apart in water and examine under the microscope. Study also prepared slides of leaf lesions in surface view or stained sections through diseased bark. OBSERVE:—

18. The color, size, septation and manner of branching. How does this mycelium compare with the germtube of the pycnospore?

19. Relation of the mycelium to the cells of the suscept; inter- or intracellular? Are haustoria developed (Hesler 1916, fig. 31).

DRAW to illustrate the character of the mycelium in the tissues.

The mycelium near the center of the lesion soon begins to form gnarls of hyphæ which rapidly develop into pycnidia, in which the pycnospores are finally produced. Study Hesler 1916, fig. 23 to learn how pycnidia are formed. DRAW.

Study sections (freehand and prepared) cut through pycnidia. OBSERVE:—

20. The dark globose structures seen in longi-section; their relation to the tissues in which embedded; the ostiole.

21. Structure of the pycnidial wall; origin and form of the conidiophores; the spores and manner of their production.

Make a detailed DRAWING of a section through a pycnidium.

When ripe these pycnospores ooze out and are disseminated by the rain. They initiate the secondary cycles.

Saprogenesis. The fungus may continue its development with the production of pycnidia in the dead portions of lesions especially on fruits and bark.

The pathogene also readily infests the dead bark of a great many plants (Hesler 1916 : 95 and 98). Pycnidia are formed on these plants and may serve as inoculum for secondary cycles.

Occasionally perithecia are formed by the saprophytic mycelium. The conditions under which they are produced is not clearly understood.

Study prepared sections through perithecia under the demonstration microscope, or Hesler 1916, fig. 18. Make DRAWINGS to show its structure.

Secondary Cycles are not different from the primary except as to time of year at which they occur.

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ENDOTHIA CANKER OF CHESTNUT

This disease has become the most destructive epiphytotic of tree diseases in this country. It is interesting in many respects. The causal fungus is an importation, and the destruction caused is typical of what may often occur when new and unsuspected pathogenes are transferred to a different flora and climate.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of this disease are primarily necrotic, but are commonly accompanied by symptoms of a hypoplastic and hyperplastic character. The lesions appear chiefly in the body and larger limbs of the tree. The symptoms are of two general sorts: the lesions or cankers themselves, and the secondary effects resulting from these cankers in uninvaded parts of the suscept. For convenience the latter are to be studied first.

Secondary Symptoms. The diseased tree may often be detected at a distance by the following symptoms which are to be studied in the photographs provided. OBSERVE:—

1. That limbs almost girdled by developing cankers, do not produce normal leaves in the spring. They are usually lighter green in color and never develop to full size. This symptom is quite noticeable during May and June.

2. That dead leaves and last year's burrs often hang to limbs which have been killed by the fungus during the summer. It is from this symptom that the disease is often called blight.

3. The dead limbs, the death of which can be traced to distinct cankers, that are of the type described below.

4. The proleptic development of suckers on trunk or limbs. Examination shows that these suckers grow from below a cankered area.

Cankers. The normal healthy bark of the chestnut remains smooth and green for several years and thus the cankers are very distinct on young growth. In the material and photographs provided, NOTE:—

5. That the cankers are at first sunken (necrotic), later they may become raised and swollen (hyperplastic).

6. That the surface of the diseased bark is always of a reddish cast, thus contrasting sharply with the green bark. (See frontispiece in Cornell Bul. 347.)

7. That in a canker of the necrotic type, the bark is usually unbroken. DRAW to show a typical necrotic canker.

8. That in a canker of the hyperplastic type the outer bark is split longitudinally because of the abnormal development of the tissues beneath. DRAW.

The **Signs** of this disease are the fruit bodies of the pathogene produced in the dead portions of the canker and the mycelial fans formed in the bark especially in the region of the cambium. Examine the cankers and OBSERVE:—

9. The numerous pimple-like structures studding the dead bark. Their relation to the lenticels.

10. The curled yellowish tendril-like threads oozing from these pimples. These are the pycnospore masses held together by a gelatinous substance which hardens on drying.

11. The pycnidium just beneath the ruptured epidermis, best seen by carefully dissecting away the epidermis with a needle; its size, shape, and color. While usually produced singly, several may sometimes be produced together and become fused so that the mass looks like a large pycnidium with several beaks or ostiola. Pycnidia are sometimes produced on bare wood in which case they are superficial. (See Anderson and Rankin 1914 : 560.)

Complete the DRAWING of the canker by showing the pycnidia in the lesion.

Make a DRAWING of the pycnidium with oozing spore mass as seen under the hand lens or binocular.

In the older lesions stromata are developed just beneath the cork layer, especially in the crevices of rough bark. Examine the material provided and OBSERVE:—

12. The orange or reddish-brown erumpent elongate stromata. Dissect away the overlying tissues and study under the hand lens.

These stromata may contain pycnidia or perithecia.

13. The shining black specks on the surface of the stromata—the ostiola or mouths of the perithecia which are buried deeply in the stroma.

DRAW to show the surface of a perithecial stroma as it appears under hand lens or binocular.

Study specimens showing the mycelial fans. OBSERVE:—

14. Their form, size and color; relation to the tissues. (See Anderson and Rankin 1914, pl. 39.)

Make a SKETCH from the specimens to show the character of these fans.

While the sunken cankered area, reddish-brown in color is the usual evidence of the disease, when the fungus attacks rough-barked portions of the tree, its effects are not noticeable externally until the stromata of the fungus break through in the cracks of the bark. This is the condition existing at the base of most trees attacked, and only the most careful scrutiny will reveal the lesion. (See illustration material.)

For the **Histologic Symptoms** of this disease study Anderson and Rankin (1914 : 589–590) and Keefer (Phytopath. 4 : 191–200).

ETIOLOGY

This disease is caused by the pyrenomycetous fungus, *Endothia parasitica* (Murrill) Andersons. It is a member of the order Sphæriales. The conidial stage, considered by itself, falls in the genus Endothiella of the Fungi Imperfecti.

Life History. Primary and secondary life cycles are not as sharply differentiated in *E. parasitica* as is usual with pyrenomycetous pathogenes. This is due to the lack of marked correlation between the periodicity of spore form production in the fungus and the seasons. Both spore forms are produced at any time throughout the year when moisture and temperature permits.

As **Primary Cycles,** will be designated only those which are initiated early in the spring, whether from ascospores or pycnospores.

Pathogeneis. This pathogene is a very active parasite once it has gained entrance to the living tissues of the suscept. It can enter however only through wounds.

We shall consider first those primary infections produced by ascospores. The ascospores are produced within the perithecia in stromata on older portions of a canker or on dead twigs and bark. In the early spring mature ascospores are discharged from the asci which have been crowded up out of the neck of the perithecium during rains (Anderson and Rankin 1914 : 580).

Soak a piece of freshly collected bark containing mature perithecia in water for ten minutes or so and placing it under the binocular watch for the discharge of the ascospores. OBSERVE:—

15. The tiny points of light popping from the ostiola of the perithecia.

Make a SKETCH to show ascospore discharge.

Hold clean dry slides at different heights above this material and determine the distance to which spores are shot in a vertical direction; also in a horizontal direction. From the slides, NOTE:—

16. That the whole contents of the ascus is ejected at once.

17. The ascospores; two-celled, hyaline, eight in each ascus. DRAW.

If shooting ascospores cannot be obtained crush a bit of the stroma in a drop of water on the slide and examine under the microscope.

Ascospores germinate as soon as discharged, if they fall in water. DRAW a germinating ascospore showing the germtubes. (See Anderson and Rankin 1914, fig. 91.)

If the germinating spore be in a wound the germtube produces a mycelium which rapidly invades the living tissues. Since the infection phenomena are identical whether resulting from ascospores or pycnospores, we will leave that for consideration until we have studied pycnospore inoculation and incubation.

Pycnospores are produced within subcortical or superficial pycnidia. Pycnidial development may be arrested at any stage by winter conditions to be resumed in early spring. Mount one of the pycnospore tendrils (spore masses) in a drop of water on a slide. OBSERVE:—

18. That the mass quickly dissolves, the spores scattering through the water giving it a milky aspect. Cover and examine under the microscope. OBSERVE:—

19. The small hyaline pycnospores. They are among the smallest spores of the fungi. DRAW.

Some conception of the large number of pycnospores produced from one canker may be obtained by noting the number observed in the mount from only a portion of one small thread. These spores, on account of their clinging together, cannot be blown about by the wind. The fact that the spore masses readily dissolve in water indicates that the chief agent of dissemination is splashing or drenching rain which carries them to wounds in the tree below the point of production. While pycnospores probably function chiefly in reinoculation of the same tree on which they are produced or on those very near by, the possibility of their being carried to distant trees by bark boring insects and insectivorus birds is not to be overlooked.

These pycnospores will not germinate in pure water but require the presence of substances from the bark in solution. The process of germination begins with enormous swelling of the minute spore. Study pycnospore-germination as shown by Anderson and Rankin (1914 : 565 and fig. 90).

DRAW to show three or more stages in spore germination.

The further activities of incubation, following germination, whether of pycnospores or ascospores consist in the development of the germtube into a mycelium in the wound. Considerable mycelium must apparently be formed in the injured tissues of the wound before infection of the living cells can be successfully accomplished.

The first evidences of infection are to be detected only after some time, in general three weeks after inoculation with pycnospores and four weeks with ascospores. Fan-like masses of mycelium are soon produced which spread in all directions through the tissues especially in the cambium (Anderson and Rankin 1914 : 563).

Examine under the microscope bits of material from the mycelial fans teased out in water and OBSERVE:—

20. The structure of the fans, branching of hyphæ and relation to each other.

DRAW to show the character of the mycelium.

After infection is accomplished and the mycelial fans begin to invade and kill living bark tissues, the canker develops rapidly. Pycnidia are produced abundantly just back of the advancing edge of the canker.

Make sections or study prepared slides of sections through the pycnidia. NOTE:—

21. The stromatic mass of mycelium of the fungus aggregated in the outer bark about the base of the fruiting structures.

22. That this stromatic layer of mycelium encases the spore-bearing cavities. The fruit bodies are said to be encased or enclosed in a stroma.

23. The irregular convoluted cavities in the stroma,—the pycnidia (Anderson and Rankin 1914, fig. 84).

24. The compact layer of pycnospores lining the entire inner wall of the pycnidium.

25. The manner of the cutting-off of the pycnospores (Anderson and Rankin 1914, fig. 86).

26. That each stroma contains a single pycnidium. The spores produced are pushed out of the ostiolum through a rupture in the bark in the long thread noted above. These spores are disseminated and initiate secondary cycles.

Make DRAWINGS to show these points.

Saprogenesis. This pathogene does not require saprogenic development for its perpetuation from year to year. But while it may produce both spore forms on dead tissues it is the pycnidial stage that first develops in the lesions. The perithecia follow later in the older long dead portions of the cankers.

The latter may therefore be looked upon as a product of saprogenesis and will be so treated here.

After a short period of pycnospore production, the stroma grows to the extent that the bark is pushed back and a cushion-like structure is raised considerably above the surface. These stroma are at first reddish in color, but later become a chocolate brown and are covered with numerous small projections or papillæ. (See Anderson and Rankin 1914, pl. 38, fig. 2.) In the cross-sections made or provided, OBSERVE:—

27. The extent of the stroma as compared with that of the pycnidial stage.

28. That the cavities here are simple flask-shaped perithecia (Anderson and Rankin 1914, pl. 40, fig. 2–3).

29. The thick, lead colored walls of the perithecia.

30. The long black necks leading from the perithecial cavity to the surface of the stroma where they open through the ostioles in the papillæ.

31. The numerous asci contained in the perithecia.

32. The ascospores in the asci; number and septation. (Anderson and Rankin 1914, fig. 89.) DRAW an ascus containing ascospores.

DRAW, showing the perithecium in longi-section. (See Anderson and Rankin 1914, fig. 85 and 88.)

The ascospores are discharged during rains into the air and carried to wound in the bark of the same or other suscepts there to initiate secondary infections. Lesions of secondary cycles produced late in the atumn mature their pycnidia or perithecia the following spring, the spores produced in these giving rise to the primary cycles again. Eventually the tree becomes covered with cankers and dies.

In the **Secondary Cycles** all the structures and phenomena of the primary cycles are exactly repeated. Secondary cycles differ in no way from the primary except in the time of year at which they are initiated.

References

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RHIZOCTONIOSE OF IRISH POTATOES

This is one of the most common and widely occurring diseases of the potato. Many other plants both wild and cultivated are subject to Rhizoctonioses but accumulating evidence indicates that the pathogenes involved are more or less distinct, biologically at least and in many cases morphologically. It is at present impossible to say just what suscepts other than the potato are liable to infection by the particular pathogene causing this disease.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of this disease are of two types, primary and secondary. The primary symptoms are necrotic and are largely confined to the underground organs—shoots, stalks, stolons and tubers. The secondary symptoms are chiefly exhibited by the tops above ground and are either hypoplastic or hyperplastic responses to the injury caused by the lesions on the stems.

Cankers are primary symptoms of the disease. Examine the specimens of diseased sprouts provided and OBSERVE:—

1. The dark colored necrotic lesions at points along the tender white underground portions of the sprouts; their form, extent and depth of penetration.

2. That many sprouts are rotted off before they reach the surface of the soil.

3. The proleptic production of new sprouts from below the rotted tip; and that these in turn may be rotted off, the process of rotting and prolepsis being often several times repeated.

4. That the cankers do not always result in a rotting off of the sprout, the shoot frequently reaching the surface and developing into a stalk. (See Morse 1914, fig. 65–66 or Rolfs 1902, pl. 3.)

Make SKETCHES to show these symptoms. Similar canker-like lesions may develop later on the stolons of young tubers or even on the roots (Morse 1914, fig. 67).

Tuber injury. Two types of tuber injury are commonly regarded as symptoms of this disease, namely cankers and russetting or cracking.

Tuber cankers are apparently not common. For a study of these see Morse (1914, fig. 70–73; read 204–207). Make SKETCHES to show such pitting or cankering of tubers.

Russetting is a very common symptom. Examine tubers provided and OBSERVE:—

5. The network of shallow cracks and checks in the surface of the tuber. Determine depth of these cracks. SKETCH to show this symptom.

These cracks may sometimes be very deep (Morse 1914, fig. 71).

Collar rot. Stalks which have survived the sprout lesions may later show lesions in the cortex at a point at or just below the surface of the soil. Examine the specimens provided (or Morse 1914, fig. 67) and OBSERVE:—

6. The girdling necrotic lesions on the fully grown stalks; color, extent and depth of the injured region. In severe cases such stalks may break over at this point. DRAW.

The secondary symptoms resulting from the canker lesions are:

Uneven stand. Early in the summer in fields in which rhizoctoniose is severe on the young sprouts, many missing hills are usually to be found. The seed piece on which sprouts are repeatedly destroyed is finally exhausted and no tops are produced or the tops of different hills are uneven in height or vigor, being either late in coming up or weak and spindling.

Rosette. Tops which do develop from cankered shoots are often weak and stunted, the leaves crowded into a rosette at the tips of the stems. (See Ohio Bul. 139, fig. 4 and Bul. 145, fig. 1.) Stalks affected later in the season also show more or less rosetting with curling and chlorosis of the new leaves at the tips.

Small potatoes. Collar rot often results in the production of large numbers of small tubers about the base of the stem. These remain small on the rotting off of the stolons. (See illustration specimens of small potatoes from a single hill or Morse (1914, fig. 69 and Rolfs 1902, pl. 9–10.)

Aërial tubers. The destruction of the cortical and phlœm tissues by collar rot lesions not infrequently results in transformation, the buds in the leaf axes developing into tuber-like storage organs to handle the carbohydrates produced by the leaves and unable to pass down to the normal storage tubers in the soil.

Study the specimens of aërial tubers provided or see Rolfs (1902, pl. 8–9) and SKETCH.

The most striking and certain diagnostic **Sign** of this disease is the presence on tubers of the sclerotia of the pathogene. Examine the tubers provided. OBSERVE:—

7. The numerous, dark brown or black sclerotia scattered over the surface.

8. Their irregular size and shape.

9. That on attempting to scrape them off, they cling very tightly, but when they come away it is evident that they are entirely superficial. DRAW a tuber to show the character and distribution of the sclerotia. These sclerotia are not usually associated with lesions but are exceedingly common on potato tubers, even from apparently healthy plants. Sclerotia may appear on the stems or roots as well as on the tubers. (See illustration specimens.)

To study the **Histologic Symptoms** make thin cross- or longi-sections through young lesions on living shoots if available. The effects on the tissues are necrotic. DETERMINE:—

10. The different types of necrotic symptoms produced. Catalogue them. Make DRAWINGS of diseased cells to show these symptoms.

ETIOLOGY

The cause of this disease, is the basidiomycetous fungus, *Corticium vagum* Berkley and Curtis, long known in its mycelial form as the sterile fungus, *Rhizoctonia solani* Kühn. The perfect stage is commonly known in Europe as *Hypochnus solani* Prill. et Del.

Life History. This pathogene presents some unusual features in its life history. True conidial bodies are wanting. Sexual spores, the basidiospores, are formed, but apparently with no regularity and only under certain very favorable conditions. Sclerotia of a very simple type are produced in great abundance and doubtless are the principal structures which serve to carry the pathogene over from one season to the next.

The **Primary Cycle** is initiated in the spring. The source of inoculum is, without much doubt, the sclerotia on overwintered suscept débris, in the soil, or on tubers.

Pathogenesis. Exactly how the primary infections are initiated does not appear to have been observed. The following studies suggest the probable phenomena involved. Tease apart, in a drop of water on the slide, one of the sclerotia from the material provided (on potato tubers or in pure culture). Examine and OBSERVE:—

11. The barrel-shaped cells in profusely branched chains which make up the sclerotium.

12. The color of the walls and the granular protoplasmic contents of many of the cells.

13. That these cells break away readily in groups of several each.

Make DRAWINGS of several of these cells together.

Many of the cells germinate like conidia. Germinate cells from sclerotia. OBSERVE:—

14. The different types of germination presented; polar and equatorial.

15. The diameter of the germtubes as compared with that of the parent cell and the number of germtubes from each.

16. The granular and vacuolate character of the contents of the germtubes.

17. The difference in the thickness and color of cell walls in germinating cells and germtubes.

DRAW several germinating cells to show the different types observed.

These barrel-shaped cells constitute the inoculum. Just how they find their way to the infection courts is not always clear. The germtubes may be capable of growing for considerable distances through the soil to the susceptible shoots which they penetrate and infect.

The vegetative mycelium, developed from these germtubes, is quite different in character from that composing the sclerotia. Mount some of the growing mycelium from the advancing margin of the agar culture provided. OBSERVE:—

18. The long parallel hyphæ, much branched and frequently anastomosing.

19. The characteristic manner of branching; the acute angle formed by the branch; the constriction of the branch at the point of origin, and the septum

laid across it a short distance above this point. These characters usually hold for the actively growing mycelium in the tissues of the suscept and serve as a diagnostic sign of the disease.

20. The colorless character of the young mycelium; the yellowing and browning with age.

Make thin longi-sections of diseased stems (or use prepared slides) and OBSERVE:—

21. The mycelium in relation to the cells of the suscept tissue; inter- or intracellular? What tissues are invaded? How does the mycelium here compare with that studied from the culture?

DRAW a portion of the section in detail to show the character of the mycelium and its relation to the cells.

Hyphæ may grow out from a diseased plant and, spreading through the soil, reach and infect nearby healthy plants. It is probable, that external mycelium and sclerotial cells, formed in the soil, may be carried in cultivation and in other ways to the neighborhood of healthy plants and so initiate secondary cycles.

Saprogenesis. The evidence seems to show that this pathogene may vegetate as mycelium in old débris of the suscept and often in the soil. At least it develops on dead plant parts and many strains appear to be almost obligate saprophytes.

A sexual form of the pathogene, the Corticium stage, is at times developed, and always on the surface of the living plant parts above the ground, i. e., on stems and leaves. Apparently it does not always occur in the life cycle. It may appear on the healthy parts above a Rhizoctonia lesion or on the stems and leaves of plants which show no such lesions or other evidences of injury. There is no indication that the fungus is ever pathogenic in this stage.

Examine the material provided or Rolfs (1904, pl. 1–3). OBSERVE:—

22. The thin, white, weft-like coating over the surface, very difficult to discern in dried or pressed material.

Scrape and mount some of this coating (or study Rolfs 1904, pl. 3); examine under the microscope and OBSERVE:—

23. That it is made up of a much branched and septate mycelium. How does it compare with the vegetative mycelium of the Rhizoctonia stage as to size, branching and septation?

24. The swollen tips of many of the branches each bearing two to four pointed sterigmata. These are the basidia.

25. Scattered through the mount, the small, hyaline, ovate basidiospores. Some half-formed or nearly matured spores still attached to the sterigmata may sometimes be found.

Make a DRAWING of the mycelium of the sexual stage, showing the fruiting structures.

The mycelium of the sexual stage appears to develop from the sclerotia on the underground parts of the suscept, or from the mycelium in the dead tissue of lesions, or from débris in the soil. It spreads upward over the surface of the living parts, without causing any direct injury, to a position where spores may be formed and readily disseminated.

The basidiospores are scattered by the wind. It is almost certain that they never function directly, infecting a healthy plant, but germinate and develop a saprophytic mycelium which may go into the sclerotial stage or at once attack healthy plants.

The development of a considerable amount of saprophytically nourished mycelium in and about the infection court seems to be a necessary prelude to an attack on living tissue.

Secondary Cycles are frequently initiated during the growing season by spreading or broken-off, vegetative mycelium. Sclerotia are readily formed and their barrel-like cells germinate at any time when conditions favor, so that they are doubtless a frequent source of secondary inoculum. So far as known, secondary cycles duplicate the primary in all details.

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TRAMETES HEART ROT

THIS timber rot is the most common heart rot in pine, hemlock, larch, firs, spruce and other conifers. It is not known to affect junipers. It is known under a variety of names such as red rot of conifers, ring shake of spruce, pecky wood rot, ring scale and conk rot. It has been estimated that this disease causes more loss than the aggregate of all the other numerous timber rots.

SYMPTOMATOLOGY

The **External Signs** of this disease are the ordinary indications of its presence in standing timber.

Studying the specimens and illustrations available, OBSERVE:—

1. The sporophores of the pathogene; their variety in form and habit (Von Schrenk 1900 : 36–40); color and external appearance.

2. That some sporophores are shelving, others resupinate.

3. That they develop from broken stubs and fissures in the bark.

Make DRAWINGS of sporophores to show the different types and their relation to the tree.

The **Internal Symptoms** are to be observed in cross- and longi-sections sawed from diseased trunks. In the specimens and illustrations provided OBSERVE:—

4. The reddish-brown discoloration of the wood in the early stages of the rot. Such timber is said to be red hearted. What tissues are affected?

5. The gray zone outside the reddish-brown wood. Especially pronounced in spruce.

6. The white areas or "pecks" present in the more advanced portions of the decayed wood.

7. That the centers of the white pecks are finally dissolved leaving holes lined with loose white fibers.

8. The size of the pockets and the amount of relatively sound wood left between them.

9. That the rotted area enlarges longitudinally in the wood much faster than transversely and often the decay involves either partially or completely a certain number of annual rings. This is the character which gives the name "ring-shake" to this wood rot.

10. The narrow black line surrounding each cavity. Read Von Schrenk (1900 : 32–33).

Make detailed DRAWINGS to show the internal symptoms as exhibited by the specimens provided.

The **Histologic Symptoms** as exhibited by decaying spruce wood may best be studied in prepared slides. Study slides of cross- and longi-sections of wood in which the pockets have not yet begun to appear. OBSERVE:—

11. That the medullary ray cells are destroyed.

12. That the elements of the spring wood are destroyed before the thicker-walled summer elements.

13. That the first noticeable change in the cell walls is the separation of the middle wall layer from the outer layer.

14. That when a hypha makes a hole through a cell wall the hole is at first cylindrical but changes by enlarging in the middle.

15. That the outer wall layer of each cell separates from the outer wall layer of the neighboring cell.

16. That, following this stage, the middle wall layer is changed into cellulose. Then there are left for a time only the outer and inner lignified layers which later also change to cellulose.

17. That the middle layer disappears first, then the outermost layer is dissolved.

18. That the only portion resisting decay is the innermost layer of the cell wall.

Make DRAWINGS to illustrate the conditions observed.

In cross- and longi-sections of spruce wood where pockets are formed, NOTE:—

19. The general appearance of the pockets under low power; their location, abundance of mycelium, and the black lines surrounding them.

20. The black line under the high power. Of what is it composed? In what tissues is it formed?

21. The fine mycelium around the margin of the pocket.

22. That the progress of the decay around the margin of the pocket proceeds as follows:—The entire wall is changed into cellulose; the elements become separated by the dissolving of the middle-lamella; the outermost wall layer then dissolves and the wall becomes thinner and thinner until only the innermost layer is left.

Make DRAWINGS to show conditions in and about the pockets.

ETIOLOGY

The cause of this wood rot is the polyporaceous fungus, *Trametes pini* (Brot.) Fries.

The **Life History** of the pathogene is similar in its essential features to the other polyporaceous fungi causing rots of the heart wood of trees. Only primary cycles are known.

The **Primary Cycles** are initiated by the basidiospores falling in wounds which afford direct communication with the heart wood.

Pathogenesis. It has been shown that the basidiospores are shot from the sterigmata with just enough force in each case to bring them to the center of the perpendicular pore from which they fall and are then caught by the wind and blown about in all directions (Buller, Researches on Fungi, **1** : 105–148). If material is available, NOTE:—

23. The clouds of spores falling from the sporophore. DRAW:—

24. The deposit of white spores on the black paper under the sporophores.

Mount some of these spores in water and under the microscope, OBSERVE:—

25. Their size and shape. DRAW.

The sources of the primary inoculum are the shelving or resupinate sporophores produced in great abundance on the outside of diseased trees. In the material provided, NOTE:—

26. That the fruit bodies are perennial. What is the evidence?

27. The black, checked upper surface of the pileus in the case of the shelving form.

28. The light reddish-brown pore layer,—the compound hymenium.

29. The small round pores making up the pore layer.

DRAW an enlarged view of the pore layer.

Make freehand sections of a fruit body provided, stain with eosin, mount and examine with the microscope. OBSERVE:—

30. The character of the mycelium forming the trama of the pileus and the substance between the pores.

31. The subhymenium and the two kinds of cells produced in the hymenium,—basidia and setæ.

32. The form of the basidium; the four spores borne on each.

If the material does not show the points mentioned above, see Von Schrenk (1900 : 40 and pl. 9, fig. 3).

Make a detailed DRAWING of a portion of the hymenium.

If the spores reach the exposed heart wood of susceptible conifers, they germinate under favorable conditions and the mycelium extends down into the trunk of the branch. The mycelium usually confines its activities to the annual rings first invaded, extending longitudinally and circumferentially in these rings much more rapidly than radially. Thus, completely decayed rings in the wood often result as the first evidence of disease. This is the ring-shake symptom. Soon, however, the mycelium invades the rings of wood adjacent to the shake and spreads inward, and outward toward the sap wood. The process outward depends upon the amount of resin present (i. e. in pine). In spruce, the fungus not only decays the heart wood and sap wood but penetrates and kills the tissue of the bark and produces small sporophores in the fissures of the same.

Saprogenesis. The pathogene may live in the wood of conifers long after the tree is dead and fallen. Sporophores also may be produced on dead trees and fallen trunks.

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IGNIARIUS HEART ROT

THIS is one of the more common of the numerous heart rot diseases of deciduous trees and may be studied as a type. The fungi causing heart rots of deciduous trees are more or less alike in their life habits and the methods for their control are similar. In this connection, see Von Schrenk and Spaulding 1909.

SYMPTOMATOLOGY

The **Signs** of this disease are the only external evidences of the malady. In the specimens provided OBSERVE:—

1. The bracket-like sporophores of the fungus, commonly called punks.

2. The point at which the sporophore emerges. Why? (See Von Schrenk and Spaulding 1909, pl. 1.)

3. The variation in the size of the sporophores. Upon what two factors does this variation depend?

4. The age of different sporophores. How determined?

5. The characteristics, shape and color, of the sporophores. Compare with other specimens on the demonstration table.

Make a DRAWING to show characteristics of the sporophore in position on the suscept.

The **Morphologic Symptoms** are all internal. In the transverse and longitudinally sawed sections of diseased wood provided. OBSERVE:—

6. The rotted wood at the center of the lesion; white or yellowish white and more or less soft and crumbly.

7. The distinct black line or concentric lines bounding the white rotted areas (Atkinson 1901, fig. 75–77).

8. The light brown or yellowish brown zone of firm hard wood just outside the outermost black line. Cause of the discoloration?

Make DRAWINGS to show the symptoms as seen in the transverse and longitudinal sections.

The **Histologic Symptoms** are highly important in wood rots since the character of the injury to the tissues has a direct relation to the commercial value of the timber. Prepared cross- and longi-sections of wood from a living maple tree are provided. These have been stained to show: lignified cell walls,—light-green (methyl green); delignified cell walls or cellulose,—brownish red (congo red); and middle lamella,—light-red (ruthenium red). The dark color of the large vigorous hyphæ when advancing into healthy tissue is natural. In the cross-sections, OBSERVE:—

9. The dense growth of the large dark-colored hyphæ making up the black line. At this point the fungus is advancing in a well defined zone toward the sap-wood. DRAW a few cells included in the black line, detailing the hyphæ.

10. The scattered areas, especially around tracheæ and in the medullary rays, where the same dark colored hyphæ are found. How do these centers of

decay originate ahead of the general advance? Illustrate your answer by two DRAWINGS, one diagrammatic, showing the relation between the black line and the advanced centers of decay, the other detailed, showing the actual cells.

11. The accumulation of green-staining material around the cells newly invaded. What does this indicate?

12. The delignification of the cell walls near the advanced centers of decay. Do the hyphæ of the fungus have to be in contact with a cell wall to cause its delignification? Proof? How is the delignification brought about? Detail answers in DRAWINGS.

13. The character of sound maple wood; elements present and thickness of the walls of different elements. Make a DRAWING from healthy maple wood showing a sector of an annual ring with all elements properly arranged and detailed. Label fully.

14. The characters of the decayed wood some distance inside the black line, noting changes in the elements. Make a DRAWING from the decayed wood of a sector of the annual ring showing the same elements as in healthy wood. Label fully and bring out all changes brought about by the decay process.

Study the longitudinal sections and OBSERVE:—

15. The mycelium advancing into healthy tissue.

16. The change brought about by the decay process inside the black line as compared with the healthy tissue outside. Make DRAWINGS of healthy and decayed maple wood, contrasting the conditions as seen in the longitudinal sections of each.

ETIOLOGY

This disease is caused by a basidiomycetous fungus, *Fomes igniarius* (Linneus) Fries, one of the perennial species of the family Polyporaceæ.

Life History. This fungus, although largely confined in its activities to the heart wood of its suscept, may be regarded as a true pathogene, because it develops only in living trees.

Primary Cycles only are known, the infections occurring once a year. With our present limited knowledge of this fungus, no sharp line can be drawn, if any exists, between its saprogenic and pathogenic activities.

The source of inoculum is the compound sporophore on the living tree. Study several of the sporophores provided and OBSERVE:—

17. The cracked and checked, hard black upper surface of the sporophores.

18. The uprolled reddish-brown margin of the last formed annual pore layer.

19. The brown surface of the pore layer.

20. The minute almost microscopic pores making up the pore layer. Study with the hand lens. The basidiospores sift forth from these pores, and are often discharged in such quantities as to be visible to the naked eye, forming a white coating on the bark and leaves beneath the sporophores.

Mount some of the spores (discharged or in crushed mounts of the pores). Examine and OBSERVE:—

21. Their form, size and color. DRAW.

Some of these spores, carried by the wind, find lodgment in dead branches or the stubs of broken-off limbs of their favorite suscepts. Here they germinate sending forth a germtube into the dead tissue. This germtube develops into a mycelium which penetrates to the wood where it spreads in all directions but most rapidly up and down, feeding and accumulating a reserve of food.

The character of this mycelium has already been determined. After a year or more, a sporophore develops from this mycelium, usually emerging from the old infection court. The lower surface develops a pore layer which is annually renewed thereafter for many years.

Examine the sporophore that has been sawed through and NOTE:—

22. That it is composed of many pore layers or strata, one below the other, each succeeding one being more extensive than the preceding. Normally one layer is formed each year. DRAW.

23. The rusty brown color of the inside of the sporophore with white flecks through it. Nature of these white flecks.

24. The length of the pores, that is, the width of the pore layer. Are the pores continuous from year to year?

25. The shape of the pore openings or mouths. Compare them, as to size and shape, with the pores of other species on the demonstration table.

Make a DRAWING of a longi-section through the sporophore, also an enlarged DRAWING to show shape and arrangement of the pore openings.

Examine free hand or prepared longi-sections of the pores or study Hartig (1878, pl. 16, fig. 1). OBSERVE:—

26. The mycelial structure of the pore layer.

27. The basidia with spores lining the inside of the pores and forming the hymenium.

28. The irregularly pointed outgrowths of some of the cells in the hymenium,—the cystidia.

Detail in a DRAWING the structures exhibited in the longi-section of a pore.

The spores sift out of these pores and are disseminated by the wind. (See Buller, Researches on fungi 1 : 105–148.)

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STIPPEN

This disease is known under a variety of names; baldwin spot, bitter pit, and fruit pit being the most commonly used. It is one of the physiogenic diseases. It occurs the world over, wherever apples are grown.

SYMPTOMATOLOGY

So far as known, lesions occur only in the fruit and appear usually as the fruit approaches maturity.

The **Morphologic Symptoms** of this malady usually develop while the fruit is still on the trees but sometimes they appear only after the fruit has been in storage for some time. Examine the specimens provided and OBSERVE:—

1. The slightly depressed areas or "pocks"; their size, shape and color.

2. Their distribution over the fruit, usually most numerous in what region? Examine illustration material and photographs. (See also McAlpine 1915–16, pl. 11).

3. The differences between these lesions and those of the New England fruit spot, specimens of which are provided.

Make a SKETCH of an affected apple to show the general character and distribution of the lesions.

Make an enlarged detailed DRAWING of a lesion of the stippen and beside it one of the New England fruit spot for comparison. Label fully.

The internal symptoms are disclosed on cutting open the affected fruit. Divide the apple lengthwise through the core. OBSERVE:—

4. The internal appearance of the lesion; color, size and shape.

5. The distribution of the lesion; relation to the surface pits and to the vascular bundles.

SKETCH the cut surface to show the form, size and distribution of the lesions as seen in this aspect.

Placing the halves together, cut the fruit across the core. STUDY:—

6. The character and distribution of the internal lesion as exhibited in this aspect, especially their relation to the vascular strands. SKETCH to show the lesions as seen in the cross-section of the fruit.

Study the individual internal lesions more critically as to:—

7. Usual form, color, texture and taste. COMPARE with the adjacent healthy tissues. Are there any indications of a bitter taste?

Histologic Symptoms. The effect on the tissues of the fruit is necrotic. Select one of the lesions next to the skin of the fruit which shows no cavity as yet. Make numerous thin sections with a sharp razor through the lesion at right angles to the surface of the fruit. Float these at once in clean water in a watch glass. Mount one of the thin but entire sections. Cover and STUDY:—

8. The diseased cells; their collapsed walls; contents and color as compared with the adjacent healthy cells. Treat the section with iodine solution and examine for starch. Where found and why present?

9. The relation of the diseased cells to the vascular cells and vessels.

DRAW to show the points brought out in 8 and 9.

ETIOLOGY

Many theories have been advanced with respect to the cause of stippen. Pathologists appear to be generally agreed that it is due to extreme variations in the water supply to the tree during the growing season. For a discussion of the various theories which have been advanced as to the cause of the disease consult the papers cited below.

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HYPOPLASTIC DISEASES

TOBACCO MOSAIC

Mosaic is one of longest known and most serious of tobacco diseases. It was the first mosaic disease of plants to be investigated. Mosaic diseases of many other plants are now known. To what extent these diseases are identical or different in different plants is not yet settled.

SYMPTOMATOLOGY

The **Morphologic Symptoms** are striking and constitute the chief evidence of the disease in the crop. Leaves, stems and flowers may exhibit the effects of the disease.

On the leaves. Examine the plants provided and locate, but do not remove, diseased leaves. Observe:—

1. The characters of a healthy leaf, its regularity of surface and margin; its size. Draw.

2. The peculiar green and yellow blotched appearance of diseased leaves. Note the blister-like elevations on the upper surface. Are they swellings or are they archings of the leaf; their dark green color. Is there any relation between the veins and the dark green areas?

3. That the yellow areas of the leaf are apparently thinner than the green islands.

4. The comparative size of healthy and diseased leaves of same age. Draw a diseased leaf on the same scale as the healthy leaf (use colored crayons). Bring out in the drawing and labeling as many of the characters as possible.

5. The distorted leaves. These may be long and ribbon-like or the lamina of the leaf may be entirely undeveloped, although the midrib is not affected. (See Allard 1914, pl. 2, fig. 1.) Draw.

6. That certain upper leaves may show a mottling (green and yellow) but without the accompanying distortions of the surface. Note how the green areas follow the veins. This is the phase that gives the names, grey-top and mottled-top, and occurs when the plant becomes infected just as the immature flower-heads appear.

On the blossoms. Examine the flowers of the plant provided, or see Allard (1914 : 5–7, pl. 3 and 4). Observe:—

7. The mottled or blotched appearance of the corollas of *N. tabacum*. Note the variations and compare with the flowers of healthy plants.

8. That some of the flowers show a depauperate growth of the corolla; the stamens and stigma extrude.

9. That the buds may be depauperate and variously misshapen.

10. That the corolla tubes of *N. paniculata* are bent and crumpled.

Make drawings of flowers and buds, both healthy and diseased, to show the various symptoms. Indicate all points, which cannot be shown in drawings, by notes in the margin.

On the plant as a whole. Compare diseased and healthy plants of different ages (Allard 1914 : 7–9, pl. 5–7). OBSERVE:—

11. The dwarfing effect of the disease.

12. That only parts of the plant may show symptoms, as certain branches, certain leaves or even parts of leaves.

13. That young growing leaves or shoots usually show the most pronounced symptoms.

14. That leaves full grown at the time of inoculation never show symptoms of the disease.

Compare the symptoms of mosaic in tomato and potato plants with those in tobacco.

To study **Histologic Symptoms** make thin free-hand sections through lesions in the leaf so as to include both dark green and light green tissues. Under the microscope OBSERVE:—

15. Difference in thickness between dark green and light green portions of the section. Determine relative thicknesses.

16. The cuboid or isodiametric form of the palisade cells in the light areas as compared with the long slender palisade cells of the normal portions of the leaf; sometimes two layers instead of one.

17. The modifications in cell contents in the diseased portions; fewer chloroplasts, and their arrangement in the cell; lighter color and disorganized condition. Read Dickson (1922 : 24–33).

Make DRAWINGS to show histologic modifications.

ETIOLOGY

That the mosaic disease is caused by living organisms of some kind seems certain from all the circumstantial evidence at hand. The fact that the virus can pass through very fine clay filters indicates that they are probably ultramicroscopic. They have never with certainty been seen and have never been grown in pure culture. The peculiar bodies to be observed in diseased cells are probably nuclear extrusions or pathologic structures of the cytoplasm (Kunkle 1921 : 6–11, pl. 5 and 9). The name *Strongyloplasma iwanowskii* Palm, has been given to the supposed organism causing the mosaic of tobacco and it is regarded as a member of the chlamydozoa, a group established by Prowazek to include the pathogens of trachoma, smallpox and similar virus diseases of men and animals. The nature of the pathogene responsible for the mosaic disease is still a matter of controversy. The student should familiarize himself with current ideas on the subject.

The peculiar pathologic structures or bodies present in diseased cells may be readily observed in the hairs (trichomes) from diseased tobacco leaves. Examine the demonstration mounts of such trichomes under the oil emersion.

Make DRAWINGS to illustrate the appearance of these bodies.

Pathogenicity Studies. The virus is readily transmitted by rubbing, injection and by insects, from diseased to healthy plants (Allard 1917).

The following simple experiments will serve to show the infectious nature of the disease.

Healthy tobacco plants will be provided. Do not touch these plants except when directed and then exactly as directed.

Experiment I. Inoculation by touching. PROCEED as follows:—

18. Thoroughly wash the hands and disinfect with the alcohol provided.

19. Squeeze and rub but do not crush, between thumb and finger, a diseased tobacco leaf; then immediately squeeze and rub with the same finger and thumb, leaves on the healthy plants. LABEL.

20. Disinfect the hands and squeeze and rub with thumb and finger (do not crush), the leaves on the healthy plants provided for checks. LABEL.

Experiment II. Inoculation with sap from diseased plants. PROCEED as follows:—

21. Thoroughly clean and rinse a mortar in sterilized distilled water. Grind three diseased tobacco leaves in a little sterilized distilled water in the mortar. The virus is thus freed from the leaf tissue. Then very thoroughly wash the hands and disinfect with the alcohol provided. Flame and cool a needle. Dip the point into the virus and prick the midrib of several young leaves of the healthy plants provided. Dip the needle each time before pricking. LABEL.

22. Thoroughly wash the hands and disinfect with alcohol. Do not touch diseased plants. Flame and cool a needle. Dip it in sterilized distilled water and prick the midrib of several young leaves of the healthy plants provided as checks. LABEL.

Experiment III. Root inoculation. PROCEED as follows:—

23. Thoroughly wash the hands and disinfect with alcohol. Do not touch the tops of the plants. Sterilize a scalpel by dipping in alcohol and flaming. Thrust the scalpel into the soil about the healthy plants provided, so as to cut some of the roots. Pour into the soil, where the roots were cut, some of the diseased leaf extract prepared in Experiment II. LABEL.

24. Thoroughly wash the hands and disinfect with alcohol. Proceed as in number 23 but use sterilized distilled water to pour on the roots, instead of the diseased leaf extract. LABEL.

Observations and Records. These plants will be kept in the greenhouse and are to be examined every two days for two weeks. Make notes on the development of the disease: (*a*) the number of days elapsing between inoculation and first evidences of infection; (*b*) character and location of first evidence of infection; (*c*) whether or not checks slow infection; (*d*) percentage of infected plants obtained in each experiment.

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POTATO MOSAIC

It is now generally recognized that there are several so-called mosaic diseases of potatoes. The most common of these in northern potato regions is the "mild mosaic," which is the disease here under consideration. This disease is now widely distributed and a serious malady of the potato (Schultz and Folsom 1923 : 46–52).

SYMPTOMATOLOGY

The **Morphologic Symptoms** of mild mosaic constitute the only basis for its identification at present, as the causal organism is unknown. These symptoms are exhibited chiefly by the foliage though the effects of the disease on the plant as a whole is to be considered.

Leaf mottling. Study the living specimens of diseased plants in pots. These have been grown from diseased tubers. OBSERVE:—

1. The irregular light green areas in the leaves which give the mottled appearance.
2. Their distribution in the blade with respect to margin, veins, etc.
3. The size, shape, and variations in degree of paleness of the light green areas.
4. Slight wrinkling or ruffling of the affected leaflets.
5. Smaller average size of affected leaflets compared with those from healthy plants.

DRAW a diseased leaf in colors.

Dwarfing. Compare diseased with healthy plants (read Schultz and Folsom 1923 : 43–54) and OBSERVE:—

6. General reduction in size of the plant as a whole; more marked in severe cases.
7. That this dwarfing is due to shortened internodes, shortened petioles and reduced size of blade in the leaflets.
8. If progeny from mosaiced and healthy hills are available, that the tubers from the diseased plants average smaller than from healthy.

Studies on **Histologic Symptoms** may be made in thin free-hand sections through strongly mottled leaflets. Read Dickson (1922 : 36–38) and compare your findings with those recorded by him.

Make DRAWING to show histologic modifications observed.

ETIOLOGY

Accumulating evidence strongly indicates a living organism to be the cause of potato mosaic but thus far no pathogene has been definitely discovered. The fact that the virus is transmissible by several artificial methods of inoculation and by aphids is the most definite indication of pathogene responsibility. The virus is also carried from one crop to the next through the tubers.

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DOWNY MILDEW OF GRAPES

This is a common disease of both wild and cultivated grapes. It is in some years very destructive to certain varieties in American vineyards but is far more destructive to cultivated grapes in Europe. It also affects the five-leaved ivy, *Psedera quinquefolia* (L) Green.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of this disease are exhibited by leaves, young canes and fruits.

On the leaves. Examine the leaves of cultivated grapes provided and OBSERVE:—

1. The location, size, color and shape of the lesions on both surfaces of the leaf.

2. Difference in the appearance of the old and young lesions, especially on the upper surface. In the early stages the lesion is a pale green to yellowish color due to the disorganization of the chlorophyl. It is only in the final stages that the tissues are killed and a typical necrotic spot is produced. (See colored illustrations.)

3. The network of fine brown lines which appear in the yellow areas (upper surface) (Gregory 1915 : 22).

4. Compare the lesions on the thin smooth leaves of the wild grape with those on the thick hairy leaves of the cultivated varieties.

Make DRAWINGS to show the characters of the lesions on the upper surface of both wild and cultivated grape leaves.

On the canes. Cane lesions may be either local or generalized. Study specimens showing limited invasion.

5. That the lesions are frequently on one side of the stem, causing it to bend or curl, the diseased area being on the outer side of the curve.

6. A slight increase in the size of the affected portion of the cane.

7. The brownish discoloration of the lesions in their advanced stages. Previous to the browning of these tissues they exhibit a distinct hydrosis observable only in fresh material.

Examine the specimens (illustration specimens) and photographs of shoots showing the generalized lesions. OBSERVE:—

8. The distinct dwarfing or hypoplasia of the shoot and its organs,—the leaves and tendrils.

Make SKETCHES of a diseased and a healthy shoot to bring out the contrasts.

Generalized lesions occur only in the young shoots which become infected as the bud begins to develop early in the spring. This general invasion of the growing tissues by the pathogene is said to be "systemic."

On the fruits. Examine the specimens and photographs provided. OBSERVE:—

9. The affected berries scattered through the bunch. In extreme cases all the fruits in the cluster may be diseased.

10. Their smaller size and brownish color. The first evidences of disease in the fruit is a blanched or livid discoloration due to disorganization of the chlorophyl as in the leaves. The advanced stages of the disease in the fruit is commonly known as "brown rot."

Compare with grapes affected with anthracnose or black rot. (See illustration specimens.)

Sometimes, particularly in the older berries, the fungus does not seem to be able to send forth the white downy fruiting structures. Such berries fail to ripen and have a whitish opaque color. This character is not well preserved in alcoholic material.

SKETCH a bunch of grapes to show the contrast between diseased and healthy berries.

The **Signs** of the disease, the downy, white coating of the lesions have doubtless already been noted. This consists of the conidial fructifications of the pathogene. Re-examine the lesions on leaves, canes and fruit. OBSERVE:—

11. The location of the conidial felt on the leaf lesions. Which surface?

12. The continuous white coating of the systemically invaded shoots.

13. The open character of the conidial felt on the diseased fruits. Why?

The conidia of the pathogene break forth from the lesions only after the yellowish discoloration has appeared. They normally develop at night and under humid conditions (Gregory 1915 : 7).

Complete the symptom DRAWINGS by adding the conidial fructifications.

The **Histologic Symptoms** of this disease are best observed in fresh material but alcoholic specimens will serve. Make thin cross-sections through a young lesion in a living leaf and OBSERVE:—

14. The disorganization and disappearance of the chlorophyl in the chloroplastids. In what tissues?

15. Effect on the nuclei of the diseased cells. On the cytoplasm.

16. Are there evidences of injury in the vascular tissues?

Prepare and study thin sections through a leaf lesion in the advanced stages of the disease. Compare conditions observed with those described by Gregory (1915 : 22).

Study thin cross-sections through a stem lesion. OBSERVE:—

17. Which tissues are affected?

18. To what is the increased size of the stem due; hypertrophy or hyperplasia?

19. The granular intercellular mycelium of the pathogene. Its relation to the increased diameter of the stem.

In thin sections through the diseased berry OBSERVE:—

20. Which tissues show evidences of injury.

21. Nature of the injury (Gregory 1915 : 23).

Make a series of DRAWINGS to show the histologic symptoms exhibited in the organs studied.

ETIOLOGY

The fungus which causes the downy mildew is a Phycomycete known as *Plasmopara viticola* (Berkley and Curtis) Berlese and DeToni. It is a native of America and was introduced into Europe about 1878 where it has since wrought great destruction.

Life History. Thanks to the extensive investigations devoted to this pathogene, our knowledge of its life history is now relatively complete.

The **Primary Cycles** are initiated shortly after growth of the suscept starts in the spring. The sources of the primary inoculum are the previously diseased overwintered leaves on the ground.

Pathogenesis. Examine bits of overwintered leaves or prepared mounts of the same. SEARCH carefully for:—

22. Rather large, globose bodies,—the oöspores, embedded in the tissues of the leaf. They are readily distinguished by the circular band-like appearance of the thick hyaline inner wall. These are resting spores which serve to carry the fungus through the winter.

These germinate during wet weather in the spring in the old leaves as they lie on the ground. The study of actual germination of these spores is attended with difficulties; therefore, study Gregory's figures and text (1912 : 235–238). OBSERVE:—

23. The large rough oöspores embedded in the disorganized leaf tissues.

24. The slender stalk,—conidiophore, sent forth from a crack in the wall of the oöspore and bearing at its tip a large egg-shaped conidium (sporangium).

COPY fig. 2, *a* and *b*. Label fully.

When mature, this conidium is readily broken off by the breeze or splashing rain drops and carried to young leaves or growing canes. Here in a drop of water, the conidium, a potential sporangium, germinates by the division of its protoplasm into (usually) 5–8 swarmspores, which are emitted through the papillate apex as shown by Gregory (1912, fig. 3).

If viable conidia are available, attempt to germinate them. (See instructions in Laboratory Technique p. 217.) Watch the process carefully.

Make DRAWINGS, either from observed germination or from Gregory's drawings, to show swarmspore formation.

These swarmspores swim about in the drops of rain or dew until they reach a stoma (on the under side of the leaf) (Gregory 1912 : 243–249). Here they cease their swimming, absorb the flagella, secrete a thin cell wall about themselves and send forth a germtube which enters the leaf through the stomatal opening (fig. 6–7). This germtube rapidly branches and spreads through the tissues between the cells.

Make thin tangential sections through the cortex of a diseased cane or berry. Mount in chloral hydrate to clear. OBSERVE:—

25. The large granular mycelium fitting so closely into the intercellular spaces that its walls are not readily distinguishable from those of the cells of the suscept; septate or non-septate?

26. The numerous small globose haustoria in the adjacent cells of the grape. They push the plasma membrane inward but do not penetrate it (Gregory 1915, pl. 2).

Make a DRAWING of the mycelium showing its structure and relation to the cells of the suscept.

This mycelium soon develops special branches which are sent forth through the stomata and branching, form the conidiophores. These conidiophores constitute the downy white growth on the surface of the lesion. Scrape some of the conidiophores from the specimen provided for this purpose. Mount in KOH solution. Examine and OBSERVE:—

27. Their color, form of branching and shape of the ultimate tips or branchlets on which the conidia (sporangia) are borne. Estimate carefully the number of conidia that may be borne on each conidiophore.

28. The egg-shaped conidia lying all about in the mount. They separate from the conidiophore very readily when mature. By which end were they attached? A mount from near the margin of a young lesion may show the immature conidia still attached to the conidiophores.

Study sections through a leaf or stem lesion. OBSERVE:—

29. The relation of the conidiophores to the suscept structures; emergence through the stomata; number from each stoma; and constriction at the stomatal opening.

Make a large DRAWING of a conidiophore in its relation to a stoma with mature conidia attached to a few of the branches.

These conidia are disseminated by the wind. They fall upon leaves, fruits and stems and initiate secondary cycles.

Saprogenesis. The mycelium within the dying leaf tissue begins the sexual formation of long lived resting spores to carry the pathogene through the winter.

The early stages in the development of these oöspores are not easily studied in the case of *Plasmopara viticola,* but may be readily observed in pure cultures of related phycomycetous fungi. Make mounts from the culture provided and OBSERVE:—

30. The large densely granular swollen tips of many of the mycelial branches,—oögonia or female organs. The cross wall cutting off some of the older ones from the mycelium.

31. The much smaller granular body, also a modified mycelial tip, applied closely to the side of the oögonium,—the antheridium or male organ. From this a fertilization tube is sent into the oögonium and a nucleus passes through to fuse with a female nucleus in the oögonium. The contents of the fertilized oögonium now round up to form a single oöspore.

DRAW to show the early stages in oöspore formation (Gregory 1915, pl. 4).

Oöspore formation may probably begin while the mycelium is still drawing nutriment from the living tissues. The oöspores are, however, not matured until after the tissues in which they are formed are dead. The oöspores ripen in the fallen leaves on the ground. Study the ripe oöspores provided. OBSERVE:—

32. The thin outer hyaline sack,—the old oögonial wall. Is there any trace of the old antheridium?

33. The uniformly thick hyaline inner wall surrounding the oily protoplasm,—the living part of the oöspore; why oily?

34. The dark colored irregularly thickened outer wall enclosing the inner wall. This consists of unused protoplasm contracted about the inner wall as a protection.

The thicker inner wall serves as a stored food-reserve for the germinating oöspores. It dissolves from within as the germinating spore develops the long conidiophore with the primary conidium (already studied).

DRAW to show the structure of a mature oöspore.

Secondary Cycles are initiated repeatedly during the season on leaves, stems and fruits by the conidia from primary and other secondary lesions. These conidia germinate, as do the primary conidia, by swarmspores. The secondary cycles repeat in all details the phenomena of the primary cycles. Only in the leaves, so far as known, are the cycles completed by the formation of oöspores.

References

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POWDERY MILDEW OF ROSES

Powdery mildew diseases affect many of the plants in greenhouse and garden. One of the most common of these is the mildew of roses. Greenhouse roses are especially liable to mildew and certain varieties of outdoor roses like the ramblers also frequently suffer with it.

SYMPTOMATOLOGY

Since the most striking features of this disease are the pathogene structures we shall study these first.

Signs. Examine the affected leaves provided (both fresh and dried). Observe:—

1. On the living greenhouse rose leaves, the powdery whitish patches of mildew on the surface. Which side? Size of the patches. To what due?

2. On the dried leaves of outdoor roses, the dense white powdery patches; much more pronounced than on greenhouse roses. Size of patches. On which side of the leaf?

3. That the mildew often runs down on the petioles. Examine some of the badly mildewed branches and clusters of ramblers (dried and pressed). Observe:—

4. That the entire surface of leaves, stems and flower clusters is coated with the mildew. Such branches sometimes arise from infected buds.

Examine under the binocular some of the leaves showing the perithecia of the fungus. These are rarely developed. Observe:—

5. The minute dark bodies in the white mycelial mats. Their size, shape and color.

The winter condition of the pathogene on the canes is very striking and characteristic. Examine some of the pieces of canes provided and observe:—

6. The dense felty white patches of mycelium often about the base of and running up on the thorns.

Symptoms. The effect on the leaves and shoots is for the most part hypoplastic. Examine the fresh leaves provided. Observe:—

7. The dwarfing and curling effect.

8. The abnormal coloration sometimes exhibited by the leaves under and about the mildew patches. Old lesions sometimes show minute black necrotic specks also.

9. The dwarfing of the shoots and flower clusters as a whole (especially noticeable on rambler branches developed from infected buds). Badly mildewed flower buds often fail to open or the hips are dwarfed and do not ripen.

Make drawings to show the signs and symptoms of this disease.

A variety of this mildew affects peaches. Study the signs and symptoms of the disease as exhibited by the peach leaves and fruits provided.

Another interesting and serious mildew of garden plants is the one which attacks the phlox. Study specimens of the powdery mildew of phlox provided and compare with that on roses.

ETIOLOGY

The cause of this disease is an ascomycetous fungus *Sphærotheca pannosa* (Wallr.) Lev. var. *rosæ* Wor. It belongs to the family Erysiphaceæ. *S. pannosa* var. *persicæ* which causes the peach mildew differs only in that it cannot attack the rose.

Life History. *Sphærotheca pannosa* var. *rosæ* is an obligate parasite. The maturing of the perithecia may be regarded as constituting its saprogenic phase.

The **Primary Cycles** are initiated in the spring on the young leaves. On the greenhouse roses there are no true primary cycles.

Pathogenesis. The sources of inoculum are the leaves on the ground on which the perithecia over-winter, infected buds, or over-wintering mycelium. The relative importance of these different sources of inoculum are not yet known.

Examine under the binocular one of the leaves bearing perithecia. OBSERVE:—

10. The minute dark colored bodies in the white mycelial mats,—the perithecia. Their shape and color.

Remove several of the perithecia, mount in a drop of water (with a little KOH) cover and as you examine them under the low power press lightly on the cover glass with the tip of a scalpel crushing the perithecia. OBSERVE:—

11. The irregular crack in the perithecial wall. Press harder until the large ascus is exposed.

12. The ascospores in the ascus. How many? Their shape, size and color. These constitute the inoculum. In nature the perithecia swell with the spring rains, the ascus protrudes and the ascospores are shot out of it.

Make a diagrammatic DRAWING of a perithecium cracked open discharging ascospores.

The mycelium in infected buds, when they start to grow, or the overwintered mycelium on the canes give rise to conidia early in the spring. These conidia may also constitute primary inoculum.

Scrape some of the conidia from a mildewed leaf, mount in water and study under the microscope. OBSERVE:—

13. Their shape, size and color.

14. Germinate some of them in a drop of water. DRAW to show conidia with some of them germinating.

The germtube of the ascospore or the conidium growing along the surface of the leaf sends haustoria into the epidermal cells of the leaf and branching forms, the mycelium.

To study the haustoria strip a bit of the epidermis from a mildewed spot, stain ten or fifteen minutes in eosin, wash, mount in water and examine under the high power. OBSERVE:—

15. The mycelial hyphæ running over the surface of the epidermis.

16. Here and there, just beneath the hyphæ inside the epidermal cells are to be seen the pink stained more or less globose haustoria. What is their function? They are the swollen tips of fine penetration tubes which arose from the surface of hyphæ on the side against the surface of the leaf.

Make a diagrammatic DRAWING (side view) to show epidermal cell in cross-section, hypha, and haustorium in proper relation to each other. (See Smith 1900, pl. 11, fig. 7–12).

Examine under the binocular the surface of a living leaf covered with the mildew. OBSERVE:—

17. The branching, spreading, septate mycelium threads over the surface of the leaf.

18. The erect chains of pearly white conidia borne on short conidiophores arising from the hyphæ.

Scrape some of them from the surface of the leaf and study under the high power. OBSERVE:—

19. The chain-like arrangement of the conidia.

20. The short conidiophores arising at right angles to the hyphæ. Where are new conidia formed?

DRAW a conidiophore showing its origin from the mycelium and its chain of conidia.

The conidia thus formed are scattered by the breeze and falling on young leaves germinate and give rise to the secondary infections.

Toward the end of the summer conidial production ceases and there may be formed on the mycelium (rather rare, apparently) sexual bodies from which perithecia are developed. (See DeBary, Morphology and Biology of the Fungi, p. 226, fig. 107.)

Saprogenesis. The leaves bearing these perithecia fall to the ground where during the autumn and early spring the perithecia mature and asci with ascospores are developed within. In the spring as we have seen these perithecia crack open, the ascus slips out and discharges its ascospores.

Secondary Cycles. These arise on the leaves, blossom clusters and shoots from conidia produced on the primary lesions. Conidiophores with conidia are produced in abundance followed sometimes by the production of perithecia just as in the primary cycles.

In some cases the mycelium spreading from infected leaves over the growing shoots invades the buds formed in the axils of the leaves giving "infected buds." Here protected by the bud scales the hyphæ live through the winter to grow and spread over the young shoot as it develops the following spring.

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POWDERY MILDEW OF CEREALS

This is a very common and sometimes serious disease of wheat, rye and other cereals. It is also to be found commonly on various wild grasses, especially species of the genus Poa.

SYMPTOMATOLOGY

The most striking evidences of this disease are the structures of the pathogene. The effects on the suscept are much less evident becoming marked only in the advanced stages of the disease. The lesions are confined largely to the leaves and leaf sheaths.

Signs. In the material provided OBSERVE:—

1. On fresh (or dried) green leaves, the white powdery patches of mycelium. On which surface? The powdery appearance is due to the great numbers of conidia which are produced on the young lesions.

2. On the older leaves the densely matted cottony masses of mycelium, more or less grey to brownish in color. As conidial production ceases there is a marked development of mycelium, forming thick cushiony mats.

3. The minute black perithecia buries in these cottony mycelial mats. Most abundant on the old dead wheat leaves (Butler 1918, fig. 57).

Make SKETCHES to show these two types of mycelial growth.

The **Symptoms** of this disease are fundamentally hypoplastic accompanied by some evidences of necrosis. In the material provided OBSERVE:—

4. The blanching or yellowing of the green leaf tissues about and under the mycelial patches; especially marked on the opposite side of the leaf.

5. In bad cases the twisting, crinkling and dwarfing of the leaves.

Show in SKETCHES any of these symptoms observed.

Where the attack is severe, there is a dwarfing of the heads or a shriveling of the maturing grains, or both. If such diseased material is available, study and compare with healthy heads and grains. Make SKETCHES to show the comparison.

Examine the illustration specimens of powdery mildews on various suscepts provided and NOTE:—

6. The marked similarity in the signs and symptoms exhibited by all of them. Note the common tendency to dwarfing in the affected organs.

ETIOLOGY

The powdery mildew of cereals and grasses is caused by *Erysiphe graminis* De Candolle, the conidial form of which is known as *Oïdium monilioides* Link.

Like all the other powdery mildew pathogenes it is an ascomycetous fungus belonging to the family, Erysiphaceæ. They develop externally upon the surfaces of their suscepts except for short haustoria sent into the epidermal cells or, in the case of one or two species, an occasional mycelial thread sent through stomata into the tissues.

Life History. This pathogene exhibits during its life cycles all of the characteristic structures of the powdery mildew fungi.

The **Primary Cycles** are initiated in the spring. The sources of inoculum are the overwintered leaves and stubble of the suscept.

Pathogenesis. Remove some of the minute black perithecia from the mycelial mats on the old overwintered wheat leaves which have been held some time under very moist conditions. Crush in a drop of water by pressing on the cover glass. OBSERVE:—

7. The large ellipsoidal ascospores forced from the perithecia; some still in the asci (Butler 1918, fig. 57). These constitute the primary inoculum. DRAW to show the form and structure of these ascospores.

When the ascospores are mature and the perithecium is thoroughly wetted, it cracks open and the ascospores are forcibly discharged. Borne by the wind, they fall upon the growing leaves of the suscept and germinate. If viable ascopores are available, study spore germination. DRAW, or COPY illustrations provided.

As soon as this germtube has established a food relation with the suscept by means of haustoria in the epidermal cells, a mycelium begins to develop, branching and spreading in all directions over the leaf surface. Examine, under the binocular microscope, one of the white mycelial colonies on the fresh green leaves provided and OBSERVE:—

8. The tangle of silver-white hyphæ with long spreading branches about the margin of the lesion.

9. The numerous erect chains of conidia borne on short conidiophores. Many of these conidia have fallen off and give the mealy appearance to the spots.

Scrape the mycelial mass from the surface of the leaf. Mount in water, cover and EXAMINE:—

10. The conidia; large ellipsoidal, flattened slightly at the ends; their thin hyaline walls and densely granular protoplasm.

11. The conidiophores; short, with a swollen base just above the point of attachment to the mycelium. This swelling is peculiar to *E. graminis*. Try to find a conidiophore with several conidia still attached. Where are new conidia formed?

12. The mycelium; very crooked and much broken in scraping from the leaf; septate or non-septate.

As the mycelium spreads over the surface of the leaf it sends minute penetration tubes through the outer wall of the epidermal cells. These enlarge within the cell to form haustoria.

To study these haustoria strip bits of the epidermis from mildew covered green leaves. Stain ten to fifteen minutes in eosin solution. Wash and mount in water. OBSERVE:—

13. The elongated elliptical pink stained body in the center of many of the long epidermal cells; the finger-like projections at either end. Why is the haustorium thus branched? Can you detect the location of the penetration tube. Examine specimens under the demonstration microscope and compare with Smith (1900, fig. 19 in pl. 11).

Make a composite diagrammatic DRAWING to show a cross-section of the epidermal cells of the leaf with haustoria, mycelium, conidiophores and conidia in normal relation to each other.

These conidia break off at the top of the chain as fast as matured and, scattered by the wind, initiate secondary cycles. Conidia continue to be produced for a time by the mycelium on the primary lesions. As the primary lesions are largely on the young or seedling leaves, the mycelium probably perishes along with the young leaf before the sexual fruit bodies can be developed. These appear later on the mycelium of the secondary cycles. There is, therefore, normally no saprogenic phase in the primary cycles.

The **Secondary Cycles** are initiated by conidia from the primary cycles.

Pathogenesis. The fungus exhibits the same conidial structures in the secondary cycles as those just studied. As the leaf tissues begin to mature, conidial production ceases, and the mycelium begins the development of perithecia. The detailed study of the development of these structures cannot well be followed out in this laboratory exercise. (See DeBary, Morphology and Biology of the Fungi, p. 226, fig. 107.)

Saprogenesis. The perithecia usually begin to appear while the leaf is still green, but do not mature their ascospores until the leaves die and have overwintered. The perithecia on the dead leaves on the ground pass the winter in an inactive condition. The rains and warm weather of spring cause the asci to mature their ascospores.

The structure of the perithecium itself may be readily studied. Examine the specimens provided, under the binocular microscope. OBSERVE:—

14. The much coarser and more densely matted mycelium; not pure white but yellowish or brownish.

15. The globose perithecia of varying sizes and colors enmeshed in the mycelial mat.

16. The short simple pale brown appendages arising from the surface of the older perithecia.

SKETCH to show the appearance under the binocular microscope.

To simulate the spring conditions, some of the leaves bearing immature perithecia have been placed in warm water for several days. They are now mature.

Remove some of the perithecia to a slide in water and cover with a cover glass. Crush by pressing firmly on the cover with the handle of a scalpel, while watching the perithecia under low magnification. What comes out of the perithecium? How many? What is the character of their contents? Does the perithecium have an ostiolum?

DRAW a diagrammatic cross-section to show a perithecium with its contents and appendages. Show the structure of the perithecial wall and mycelial attachment.

With the maturity and discharge of the ascospores, the secondary cycles are completed.

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POWDERY MILDEW OF THE APPLE

Most fruit trees are subject to some powdery mildew disease. The powdery mildew of the apple will serve as a type as they are all rather similar in character. While not uncommon in Eastern apple orchards it is in the Northwest where the disease becomes of real economic importance.

SYMPTOMATOLOGY

Since the most striking evidences of the disease are the pathogene structures we shall study these first.

Signs. The growing leaves, twigs, blossom clusters and shoots exhibit the signs of this disease most strikingly. Examine the growing young apple trees* or twigs provided. Observe:—

1. The white powdery coating of the leaves. Which side? Of what composed? Note that petioles and twigs are also coated.

2. The tendency to more abundant development of the powdery coating along the veins. Draw to show the character and pattern of the mildew on the leaf.

Examine the watersprouts collected in the autumn. Observe:—

3. The thick felty mycelial coating. Its dirty white or brownish tinge. Compare carefully with that on the young growing leaves and shoots (Fisher 1920, fig. 2).

4. The minute black perithecia, more or less embedded in the mycelial mats. Study under the binocular microscope.

Draw to show this winter condition.

Symptoms. The hypoplastic character of the symptoms of this disease are usually marked. Compare the diseased plants before you with healthy ones. (See also Ballard 1914, pl. 4, 5 and 6; Fisher 1918, pl. 1 and 2; and Fisher 1920, fig. 1.) Observe:—

5. The marked dwarfing, curling and rolling of the leaves.

Make sketches to show the different types of leaf malformation.

6. The dwarfing or shortening of affected shoots as shown by specimens and illustrations cited above.

7. Increased diameter of affected twigs.

8. More or less defoliation. Severely injured twigs often winter kill.

Make sketches to show these effects on the twigs.

The flower clusters also exhibit certain well marked symptoms. Study Fisher (1918, pl. 3 and Fisher 1920, fig. 3). Observe:—

9. The general dwarfing effect on flowers and leaves of the spur.

10. The malformation of leaves and flower petals. Virescence of the petals is not uncommon. Mildewed flowers usually fail to set fruit.

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* Seedling apples or year old buds forced in the greenhouse usually show abundant development of mildew.

11. The slowing up of blossom development (Fisher 1918, pl. 3).

Make a DRAWING to show the effects on blossom clusters.

The growing fruits may also be attacked and exhibit certain characteristic symptoms as a result. Study Fisher (1918, pl. 1, fig. 2 and Fisher, 1920, fig. 4–5). OBSERVE:—

12. A russetting of the surface of the apple. How can this be explained? DRAW.

ETIOLOGY

The powdery mildew disease is caused by the ascomycetous fungus *Podosphæra leucotricha* (Ellis and Everhart) Salmon. Another species of Podosphæra, *P. oxycanthæ* (DC.) DeBary, is also said to occur on the apple but is certainly not common.

Life History. Like all species of the Erysiphaceæ this pathogene is a strictly obligate parasite. Its saprogenic phase consists in the overwintering and maturation of the perithecia.

The **Primary Cycles** are initiated in the spring on the new leaves as they develop.

Pathogenesis. The sources of inoculum are two, namely, the shoots and twigs bearing overwintered perithecia and the infected buds.

From one of the watersprouts provided, remove with the scalpel, several of the perithecia to a drop of water containing a little KOH on a slide. Cover and while examining under the low power crush the perithecia by gently pressing on the cover glass with the scalpel. OBSERVE:—

13. The irregular crack in the perithecium from which the ascus is forced out.

14. The large ellipsoidal ascospores usually remaining within the ascus, number; color; contents.

15. The thin place in the wall of the ascus at the apex. At maturity this dissolves as the perithecium cracks open and the ascospores are forcibly ejected into the air.

Make a DRAWING of a cracked perithecium with protruding ascus discharging spores.

Borne by the breeze, these ascospores fall upon young shoots or leaves of the apple and germinate. The germtube grows out over the surface and sends a haustorium into an epidermal cell. From this germtube the branching mycelium develops. Although investigators (Fisher 1918 : 5–6) hold the ascospores of *P. leucotricha* to be of little or no importance in initiating new infections in the Northwest, it is highly probable that they do so function under other conditions. That apparently is their normal rôle in the case of many other Erysiphaceæ.

The other and usual source of inoculum appears to be buds formed last season which were invaded and infected by the mycelium of the pathogene. Such infected buds usually start growth a week or two after healthy buds have begun to develop. As these infected buds develop the mycelium of the pathogene in them also grows and spreads over the unfolding leaves and twig and blossom clusters (if it be a blossom bud). The conidia which are promptly produced in abundance constitute the primary inoculum for the infection of the leaves developing from healthy buds to which they are carried by the wind. Since these "infected buds" are difficult to obtain, conidia from some other source may be substituted for the study of the primary infection.

16. Remove some of the conidia from the living apple leaves provided and set them to germinate in water. Study and SKETCH later.

The germtube produced from the conidium develops into the mycelium which branching and spreading over the surface of the leaf sends haustoria into the epidermal cells. Slice or peel off bits of the mildew covered epidermis from a living leaf (use a sharp razor). Stain for ten minutes or so in eosin, wash away the stain and mount in a drop of water. Under the low power examine the edges of the slices for places where epidermal cells only show. Put on the high power and LOCATE:—

17. The slender septate mycelium.

18. In some of the epidermal cells across which the hyphæ grow, rather large pink stained globose bodies,—the haustoria. The bulge in the hypha from which the haustorium arose. The slender penetration tube uniting hypha and haustorium—this appears as a minute opening seen through the hypha and near the center of the haustorium. It is seen in cross section. (See Smith 1900, pl. 11, fig. 12.)

DRAW to show top view of hypha, penetration tube, haustorium and epidermal cell.

From the mycelium there soon arise numerous conidiophores. Examine the surface of a living mildewed leaf under the binocular. OBSERVE:—

19. The web-like hyphæ on the surface of the leaf with numerous erect branches bearing bead-like chains of conidia. (Compare Ballard 1914, fig. 2.)

Scrape some of the mildew from the leaf. Mount in water, cover and study under high power. MAKE OUT:—

20. The structure of the conidiophores and their origin from the mycelium. At what point are new conidia produced?

21. The individual conidia, form, color, contents.

Make a diagrammatic DRAWING of a conidiophore with conidia, showing its relation to the mycelium, haustoria and epidermal cells of the leaf.

It is these conidia which scattered by the breeze fall upon other leaves and so give rise to the secondary cycles.

With the falling of the leaves the mycelium and conidia remaining thereon die. It is only on the twigs that the fungus can pass the winter. This it does as we have seen in one of two ways: (1) The mycelium which has grown down the petioles onto the growing twig may penetrate between the bud scales of the developing buds in the axials of the leaves where it remains dormant until the following spring; or (2) perithecia may develop on the mycelial mats on the twigs. The detailed study of the development of the sexual bodies and the formation of the perithecium cannot well be undertaken in this exercise (see DeBary, Morphology and Biology of the Fungi, p. 226, fig. 107).

These perithecia are formed during the summer and autumn but do not mature until the following spring. The mycelium in the mat on which they are formed probably die during the winter.

Saprogenesis. The maturation and discharge of the ascospores from the perithecium may be regarded as saprogenesis since it takes place independently of the living suscept (though usually upon it) and at the expense of foods stored in the perithecium.

Mount and study some of the mature perithecia. OBSERVE:—

22. The cellular character of the perithecial wall.

23. The long slender appendages adorning the top of the fruit body. These are characteristic of this species.

24. The short crooked and branched appendages at the base. These serve to anchor the perithecium to its substratum.

25. On crushing the perithecium, the single large ascus containing the asci. The perithecia of most Erysiphaceæ contain more than one ascus. Podosphæra species always contain but one.

Make a diagrammatic DRAWING of a perithecium in longitudinal section.

The **Secondary Cycles** are, as pointed out above, initiated by the conidia from the primary lesions. They normally repeat in all details the phenomena and structures exhibited in primary cycles. Conidia from secondary lesions may repeatedly initiate other secondary cycles during the season.

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APPLE SCAB

Of all the diseases of the apple, this is the most common and best known to the growers. It is the one fungous disease for which they spray or dust. It is world wide, occurring practically wherever the apple is grown. The scab of the pear which is very similar in its symptoms to the apple scab is caused by a distinct but closely related pathogene.

SYMPTOMATOLOGY

Morphologic Symptoms of all three types may be exhibited by this disease. The predominating symptoms are, however, hypoplastic. Lesions occur on leaves, flowers, fruits and the current year's twig growth.

Leaf blotch. The leaf lesions present a typical blotch symptom. The injury is usually superficial, the outstanding feature of the symptom picture being the dark colored mycelium of the pathogene. The first evidence of the disease in the spring is on the unfolding leaves. The blotches usually appear first on the lower surface, but later new ones appear on the upper side of the leaf. Examine the leaves provided and OBSERVE:—

1. The size, form and character of the blotch. The radiating character of the lesion. To what due?

2. The character of the injury to the leaf. Does the injury show on the surface opposite the blotch?

3. The difference in the character of the upper and under surface of the leaf itself; and of the lesions on the two surfaces.

4. The variations in the character of the scab lesions on different leaves. (Compare Wallace 1913, pl. 1.) Badly scabbed young leaves are often dwarfed and distorted.

Make DRAWINGS to show the characters of the scab lesions on the upper and under surfaces of the leaf.

Sometimes, the tissues beneath the blotch die producing a characteristic spot. This commonly results from the penetration of toxic substances from fungicides applied after the blotch has formed and destroyed the protecting cuticle of the leaf. Examine the specimens provided and OBSERVE:—

5. The necrotic lesions, their color, shape and relation to the original blotched area.

6. The radiating mycelial hyphæ on the surface of the necrotic area in every case.

DRAW to show scab lesion spray injury. Scab lesions may also occur on leaf petioles (see Cunningham, N. Zealand Dept. Agr. Bul. n. ser. 100, fig. 2). Such lesions may cause abscission resulting in defoliation.

Blossom blight. Lesions may appear on the pedicle and calyx of the flower before the petals fall and may be severe enough to prevent the setting of the fruits. (See Wallace 1913, cover picture and pl. 7.) In the material provided, OBSERVE:—

7. The location, form and character of the scab lesions and the effect on the flower.

On our Pacific coast where cool rainy weather sometimes prolongs the blooming period infection of the petals may also occur. Examine the illustration specimens provided.

Make DRAWINGS to show blossom blight lesions.

Twig scab. Scab lesions on the current year's twig growth is rather uncommon except in certain localities especially favorable to the pathogene, as for example northeastern United States, Nova Scotia, the eastern shore of Maryland, England and New Zealand. The twigs of certain varieties seem to be more liable to twig lesions than others. In the material or photographs provided, OBSERVE:—

8. The rough blistered character of the lesions, confined to the growth of the current year. (See also Cunningham, N. Zealand Dept. Agr. Bul. 100, fig. 6–7.) DRAW.

Fruit scab. Where the infection of the calyx is not severe enough to prevent the fruit from setting the lesion develops into a typical scab spot as the apple grows. Secondary lesions often appear as the fruit grows older. These become very evident as the season advances. In the young apples provided, OBSERVE:—

9. The black scabs. Their form, size, and effect on the fruit. To what region on the apple are they largely confined? Why?

10. The felty black center of the scab in old lesions. In some cases, this felt has disappeared and the center is hard, corky, reddish-brown and often cracked.

11. The papery rim or border, best seen in the younger lesions. This consists of the cuticle of the apple which has been loosened by the fungus as it spreads from the center of the infected area.

Make DRAWINGS to show the typical scab lesions on the fruit.

Sometimes these lesions cause a dwarfing of the apple on the affected side, in other cases they cause prominent swellings, especially on Ben Davis. (See demonstration specimens.)

Show these effects in SKETCHES.

The **Histologic Symptoms** of the scab are most advantageously studied in free-hand sections through lesions from living young fruit. If material is available make sections and OBSERVE:—

12. The rupture and dissolution of the cuticle.

13. The dark stroma-like mass of fungous hyphæ resting on the epidermal cells often crushing them out.

14. The suberization of the walls of the cells just beneath the epidermis, indicated by the brown coloration of the walls.

DRAW to show histologic changes.

ETIOLOGY

The apple scab is caused by the conidial stage, *Fusicladium dendriticum* (Wallroth.) Fuckel, of an ascomycetous fungus known as *Venturia inæqualis*

(Cooke) Winter (=*V.* pomi (Fries) Winter). It belongs to that group of the ascomycetes known as Pyrenomycetes which have their asci enclosed in a more or less globose fruit body called a perithecium.

Life History. It is in the conidial stage that this fungus exhibits its pathogenic nature. It lives superficially on the suscept or nearly so, simply dissolving away the cuticle or upper part of the epidermal cells, and applying its mycelium closely to the tissues beneath.

The **Primary Cycles** are initiated by ascospores from perithecia in old leaves on the ground.

Pathogenesis. Crush, in KOH solution, a bit of the old leaf containing perithecia, examine and OBSERVE:—

15. The two-celled olivaceous ascospores. DRAW. These ascospores are shot from the ascus which protrudes when ripe through the ostiolum of the perithecium. Ascospores are dischared only during rains in spring. They are carried to the young leaves just emerging from the buds. Here they germinate. Study Wallace (1913, pl. 9–10) and OBSERVE:—

16. That but one cell of the ascospore gives rise to a germtube. Which cell? DRAW to show three stages in the development of the germtube. This germtube pierces the cuticle of the leaf or young fruit and initiates the scab lesion. Again examine the blotches on the leaf and with the hand lens or binocular MAKE OUT:—

17. The branched mycelial threads. Why do they radiate from a common center? Make an enlarged DRAWING of a lesion to show this habit.

Study the mycelium, as shown in the prepared slides of apple leaves which have been cooked in KOH solution and the epidermis, bearing the fungus, peeled off. OBSERVE:—

18. The form, size and septation of the mycelium. Its color and method of branching. No haustoria are sent into the suscept cells and the mycelium does not at this stage penetrate beyond the epidermal cells.

19. That the conidiophores arise in clusters or singly from this spreading mycelium. Note their form, length and color.

20. The conidia lying about, which have been broken off from the conidiophores; their form, size and color and point where they were attached to the conidiophore. Several conidia may be produced from near the same point on a conidiophore. How? See if you can find a conidiophore which shows this.

Make DRAWINGS to show the mycelium, conidiophores and conidia in their proper relations to each other and to the suscept.

Scrape some of the conidia from the scab on an apple. Mount and study. Compare them with those found in the prepared slide. They are sometimes 1-septate.

Make DRAWINGS to show the variations in size, form and septation of the conidia.

Saprogenesis. The mycelium in the primary lesions on the leaves continues to produce conidia until the leaves fall. The fungus, which has up to that time lived practically on the surface now sends new branches of the mycelium throughout the dead tissues of the leaf. From this mycelium are formed the globose perithecia. They are formed just beneath the epidermis of the lower or upper surface of the leaf, being most abundant near the surface facing upward as the leaf lies on the ground. Examine the old apple leaves provided and OBSERVE:—

21. The evidence of the old scab blotches of the summer.

22. With the hand lens the pimple-like perithecia scattered over both surfaces of the leaf just beneath the epidermis. Note the color, size, distribution, relation to the old lesions and relative abundance on the two sides of the leaf.

Make a DRAWING of a portion of the leaf to show the perithecia as seen with the hand lens or binocular.

With the scalpel, cut from the leaf a small square of tissue showing an abundance of the perithecia. Place between pieces of pith and with a razor make free-hand sections through the perithecia. Mount and study. OBSERVE:—

23. The shrunken dead condition of the leaf tissues.

24. The embedded perithecia.

25. The mycelium of the fungus throughout the tissues of the leaf; its form and character.

26. The structure of the perithecium. (Best made out in the stained sections.) The walls, their relation to the leaf tissues, form, size, mouth or ostiolum and asci, with ascospores. How many ascospores in an ascus?

Make a DRAWING to show the structure, contents of the perithecium and its relation to the leaf tissues (Wallace 1913, fig. 183). The perithecia begin to develop in the autumn but do not mature their ascospores until spring at the time when the apple leaves and blossoms are unfolding.

Secondary Cycles are initiated by the conidia from the lesions on leaves and fruits on the tree. Aside from their conidial origin, the secondary cycles which are repeatedly initiated throughout the season, do not differ from the primary.

27. Germinate conidia from a lesion on the fruit. DRAW.

The fungus forms on the fruit a thick stroma-like growth from the upper surface of which arise the short conidiophores bearing the conidia. In order to study this structure and the relation of the pathogene to the tissue of the fruit, make thin cross-sections through the scabs on the half-grown fruits. OBSERVE:—

28. The thick stroma-like mass of the mycelium. Its pseudoparenchymatous structure; thickest toward the center of the lesion.

29. The short conidiophores with conidia. Find conidiophores that show different stages in their formation and development.

Make a DRAWING to show the structures observed.

References

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ERGOT OF RYE

This disease is one of the earliest known and most frequently studied ascomycetous diseases of cereals, due doubtless to the poisonous effects of the sclerotia of the pathogene on man and beast, rather than to the reduction of crop yields which it causes. The suscept of greatest economic importance is rye though it is at times very severe in wheat and certain meadow grasses.

SYMPTOMATOLOGY

The **Signs** of this disease are the most striking evidences of its presence in an affected crop. These signs are of two types, those of the honeydew stage and those of the ergot stage.

Honeydew stage. Examine the young heads of rye provided and OBSERVE:—

1. Smudgy traces of the now dried honeydew. This exudate is often difficult to see in dried specimens. In fresh material it is a milky fluid, sweet and sticky. It is often copious, covering the entire head and running down the culm. As it dries adhering dust gives the affected portions the smudgy appearance.

Ergot stage. In the mature heads provided OBSERVE:—

2. The black spur-like bodies projecting here and there from between the glumes,—the ergots; average number from each head. Examine illustration specimens of other ergot-infested grains and grasses.

DRAW a head of rye and a head of one other suscept showing the ergots.

Examine the loose ergots provided. NOTE:—

3. The variation in size and shape; consistency; the surface, often checked and cracked. These ergots are sclerotia of the pathogene, developed in place of the kernel from the structures of the honeydew stage, the shrunken remnant of which may often be observed still clinging to the tip of the sclerotium.

4. On breaking one open, the light colored interior. These ergots are poisonous.

Make an enlarged detailed DRAWING of two or more of the sclerotia.

The **Morphologic Symptoms** are exhibited chiefly by the diseased ovaries. In preserved (alcoholic) material, taken at the honeydew stage, or in soaked heads which have been preserved dry, dissect away the glumes and OBSERVE:—

5. The whitish kernels with corrugated surfaces. Compare with normal kernels from the same or healthy heads of the same age, as to size and color (Weniger 1924, fig. 4).

6. The glumes about the diseased kernels. Are they diseased? Compare with healthy.

It is from these diseased kernels that the honeydew oozes, but in the dried specimens, there is evident only the whitish covering of the diseased kernels.

Make a DRAWING of the ovaries from a diseased and a healthy spikelet to bring out these characters.

The **Histologic Symptoms** may be investigated by a comparative study of thin longi-sections through diseased and healthy ovaries from alcoholic material.

Illustrate your findings in carefully made DRAWINGS.

ETIOLOGY

The ergot is produced by the ascomycetous fungus, *Claviceps purpurea* (Fries) Tulasne, the conidial or honeydew stage of which was given the name, *Sphacelia segetum* Léveillé.

Life History. Several careful researches have resulted in rather complete knowledge of the life history of this pathogene. Primary and secondary cycles with sharply marked pathogenesis and saprogenesis occur.

The **Primary Cycles** are initiated in early summer at the time the rye or other grass suscepts begin to head out.

Pathogenesis. Ascospores which are produced in the spring constitute the inoculum for the primary cycles. From the ergots (sclerotia), which overwinter on the ground, there develop in the spring, stalked fruit bodies. In the heads of these are developed numerous perithecia containing the asci with long slender ascospores. Examine the specimens provided. OBSERVE:—

7. The little pimple-like dots scattered thickly over the globose head of the fruit body,—the ostiola or mouths of the perithecia.

From these ostiola the long thread-like ascospores are shot into the air, and, carried by air-currents, lodge in the infection courts,—the open blossoms of the rye or other grasses.

A bit of material from a mature stroma will be provided. (Ask instructor.) Crush, cover and study. OBSERVE:—

8. The long slender ascospores; some floating free, others still in the asci; continuous or septate? DRAW several ascospores. (Keep this slide for further study later.)

These germinate within the blossom. Examine photograph or DeBary (1887, fig. 109) for germinating ascospores. NOTE:—

9. That upon germination the entire spore at once becomes virtually a part of the mycelium. COPY.

The branching germtubes quickly penetrate the young ovary and growing throughout its tissues, develop the sphacelia or conidial form of the fungus.

Study cross-sections (freehand or prepared) of the upper end of the ovary (Sphacelia stage). Examine with the high power. OBSERVE:—

10. The numerous irregular cavities lined with erect conidiophores bearing minute ovate conidia. Note the angular cells of the ovary.

DRAW a small portion of the hymenium with underlying ovary tissue, showing the conidia and the manner in which they are borne.

The honeydew consists of a sweet fluid exuded by the pathogene, in which great numbers of these conidia find their way to the surface of the spikelet.

Insects attracted by the honeydew serve as inoculating agents in starting the secondary cycles.

Very soon the mycelium in the basal portion of the ovary begins to develop the sclerotium, which, as it grows and develops, replaces the ovary and pushes forth as a long black spur with the shriveled Sphacelia structures at its tip. It is at first light colored and inclined to purplish but soon turns black. (See illustration specimen.)

Examine the photographs provided. NOTE:—

11. That while the sclerotium usually replaces the entire ovary, this is not always the case. COPY from the illustrations to show this.

Make very thin cross-sections of one of the mature sclerotia (soaked) and study under the high power. OBSERVE:—

12. Its pseudoparenchymatous structure.

13. The thick walls and small lumina of the hyphæ composing the sclerotium. Are there any traces of ovary tissues?

14. The dark color of the outer coat or rind; number of cells thick; the white color of the medulla.

Detail in a DRAWING the structure of the sclerotium.

Saprogenesis. These sclerotia, when mature, fall to the ground or find their way at threshing into bins along with the grains. With these they may be sown and find their way to the soil. Here they remain dormant until spring, when the fungus again becomes active. At points beneath the rind, growth centers may be set up from which are developed slender stalks with globose tops. These are developed at the expense of the food materials stored in the cell walls of the sclerotial medulla.

Examine the so-called germinating sclerotia provided. OBSERVE:—

15. The small, capitate bodies arising from the sclerotium,—the stromata. Note the comparative size of the stem and the head. Examine the stromata carefully with the hand lens, noting the small punctations:—the ostiola of the perithecia.

Make a DRAWING to bring out all the external characters of the stromata and their relation to the sclerotium.

Examine the prepared slides of cross-sections of the stromata provided. OBSERVE:—

16. The ovate, acuminate cavities,—perithecia. Note the compact pseudoparenchymatous wall of the stroma.

17. The location of the perithecia about the periphery of the stroma.

18. The opening at the top,—the ostiolum. Note that it protrudes. Make an outline DRAWING of the cross-section of the entire stroma.

19. The asci arising from the bottom of the perithecium.

20. Within the asci, the bundle of eight thread-like spores. Supplement at this point with the crushed mount prepared under number 8. Stain by running a little methyl blue under the cover glass.

Make an enlarged DRAWING of a perithecium containing the asci and of a single enlarged ascus with the ascospores.

Secondary Cycles initiated by the conidia of the Sphacelia stage, multiply and spread the pathogene during the blossoming period of the suscept.

The Sphacelia stage, followed by the development of the ergot, is produced as in the primary cycles.

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ONION SMUT

This disease is prevalent only in the cooler onion sections of North America. It occurs also in England. It frequently causes losses of 50 per cent. or more. Because of its striking symptoms it has long been known to onion growers and has been the subject of study by scientists for thirty years.

SYMPTOMATOLOGY

The diagnostic evidence of this disease is the presence of the black spore masses in the tissues of the leaves and bulbs. Examine the diseased seedlings provided, holding them to the light. OBSERVE:—

1. The opaque masses within the tissues of the leaves. These are the sori of the pathogene.

2. The slight swelling in the leaf at the point where the sorus occurs.

3. On cutting across the leaf through the opaque spots, the black spore mass within.

4. The ruptured epidermis and exposed spore mass in some specimens.

5. The withered tip of badly diseased leaves.

Make a DRAWING of a diseased seedling.

Examine one of the larger diseased onions. OBSERVE:—

6. That each badly diseased leaf has turned brown and withered.

7. That the black lesions have now broken open, forming long black open sori from which a sooty-like mass is sifting.

Cut across one of the mature diseased bulbs. OBSERVE:—

8. The large black sori embedded in the fleshy scales. How may layers of bulb scales are affected?

Make DRAWINGS to show symptoms in the mature bulbs.

Badly diseased seedlings are often killed before bulb formation begins and even when bulbs do form they are smaller than healthy ones. Their tops die early and they are frequently invaded by other soil fungi which rot them.

ETIOLOGY

The pathogene responsible for this disease is *Urocystis cepulæ* Frost, a basidiomycetous fungus of the order Ustilaginales. There are two families in this order, the Ustilaginaceæ and the Tilletiaceæ. To the former belong those organisms which produce the loose smuts of wheat, oats and corn, while to the latter belong the pathogenes like those producing the stinking smut of wheat, and the onion smut.

The **Life History** of this smut pathogene is strikingly different from that of most cereal smuts in that it is a soil inhabiting organism and not intimately associated with the seed of its suscept.

Primary Cycles only occur and the pathogene exhibits a well marked saprogenic stage.

Pathogenesis. The chlamydospores, set free by the disintegration of the diseased onions, germinate more or less promptly producing a saprophytic mycelium which thus established in the soil constitutes the inoculum. When onion seed are planted in infested soil, invasion by the mycelial hyphæ occurs from three to seventeen days after germination begins. Anderson (1921 : 123) holds that the cotyledon only, serves as infection courts but Walker and Jones (1921 : 239) believe that the basal portion of the first leaf may for a time also be invaded.

Since special technique is required for a study of penetration, read Anderson (1921 : 123–125). Copy one of Anderson's DRAWINGS in fig. 4. Label fully.

To study the mycelium in the tissues select a diseased seedling in the cotyledon stage and make thin longi-sections through the basal portion just above the first roots. Stain with eosin for some time, wash and examine with the high power. (Prepared slides may also be used.) OBSERVE:—

9. The granular mycelium. Inter- or intracellular? Septate or nonseptate? In what tissues does it occur? Search for haustoria.

Make a detailed DRAWING of a portion of the invaded tissues showing relation of the mycelium to the cells of the onion.

The developing mycelium soon masses at certain points to form the chlamydospores. Study cross-sections through a sorus. OBSERVE:—

10. The large compound spores packed together in the sorus. To understand the manner of their formation study Anderson's paper (1921 : 128–131).

Saprogenesis begins with the maturing of the chlamydospores. Mount some of the mature chlamydospores from an old sorus. OBSERVE:—

11. Their form, size and structure. DRAW. If fresh material is available germinate some of the chlamydospores on onion decoction agar using the method described by Anderson (1921 : 106). If fresh spores are not available study Anderson's drawings (fig. 1).

Make DRAWINGS of germinating spores at different stages.

The chlamydospores are commonly distributed in or on diseased onion sets, but there is some evidence that they may also become attached to the onion seed in harvesting and so be transported to new areas. Seedlings of other plants such as celery grown in infested soil may easily serve as bearers of the saprophytic mycelium in adhering soil.

Pathogenicity Experiments. *Experiment 1.* Sow onion seed in infested soil in greenhouse flats. Place the flats in a cool house where the temperature does not exceed 25° C. Remove some of the seedlings every day or two after they begin to come up and examine for evidences of infection. Record your observations.

Experiment 2.—To observe the effect of temperature on infection carry a flat sown as above in a warm greenhouse (27° C. or higher) and compare the percentage of infected plants with that obtained in the cool house.

Experiment 3.—To determine the age at which the onions become immune to invasion, grow a flat of seedling in disease free soil. Beginning as soon as they are up, transplant, 10 or more every other day for three weeks to infested soil in the cool house. Pull and examine each lot at the end of five or six weeks and record the number smutted in each lot.

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LOOSE SMUT OF WHEAT

THIS disease is exceedingly common and often destructive in the wheat fields of United States and Canada. It is readily distinguished from the stinking smut. The latter, while not uncommon in the east, is much more general and destructive than loose smut in the great wheat lands of the west. While the loose smuts of wheat and oats are much alike in general appearance, they differ strikingly in certain features of the life history of their respective pathogenes.

SYMPTOMATOLOGY

The **Signs** of this disease are its most striking symptomatological features. They are to be observed only at flowering time.

Compare a diseased and a healthy head of wheat. OBSERVE:—

1. The powdery black mass of spores which characterize the diseased head. DRAW.

The **Morphologic Symptoms** are best studied in the field but some effects of the pathogene may be observed in preserved material. OBSERVE:—

2. The general effect of the disease on the form and appearance of the head. Does it affect the rachis as to length and size; length of internodes? DRAW both diseased and healthy heads.

3. The effect on the individual spikelets. (See Freeman and Johnson 1909, pl. 4–5.) Enumerate the modifications. Determine this by carefully dissecting out (in water) the parts of the healthy spikelets and flowers. DRAW. Dissect and DRAW the parts of the diseased spikelet.

4. The naked rachides from which the spore masses have disappeared. DRAW.

In the field it may be OBSERVED:—

5. That the culms of a diseased plant at first grow more rapidly and outstrip those of the healthy plant. Eventually, after spore dispersal, the healthy culms push up above the diseased ones.

6. That (according to McAlpine 1910 : 86) growing culms affected with smut are generally of a purple tint.

7. That both diseased and healthy heads occur commonly in the same stool.

8. That the upper spikelets of the head may sometimes escape invasion and develop normally. (Freeman and Johnson 1909, pl. 5c.)

9. That sori occasionally develop on the upper leaves and even the culms (Butler 1918 : 165).

ETIOLOGY

The loose smut of wheat is caused by the basidiomycetous pathogene, *Ustilago tritici* (Persoon) Jensen. It is a member of the order of primitive basidiomycetes, the Ustilaginales, commonly known as the smut fungi. There are two families in this order, the Ustilaginaceæ to which *U. tritici* belongs and the Tilletiaceæ to which belong the stinking smut pathogenes, *Tilletia tritici* (Bjerk.) Wint. and *Tilletia fœtens* (B. and C.) Trel.

Life History. While the pathogene causing this disease has long been known, it is but recently that its life history has been completely understood. It differs (along with *U. nuda* (Jens.) Kell. and Sw. in barley) from most of the other smut fungi whose life histories are known, in that infection occurs through the stigmatic surfaces of the pistil at flowering time. There are no secondary cycles.

The **Primary Cycle** requires a full year for its completion. There is no saprogenesis, the pathogene remaining in continuous association with the living suscept from the moment of inoculation to the maturity of the chlamydospores.

Pathogenesis. The black smutted heads emerging from the upper leaf sheaths at flowering time constitute the sources of inoculum.

Mount some of the sooty mass in a drop of KOH solution, cover and examine with the high power. OBSERVE:—

10. The numerous brown globose bodies scattered through the mount,—the chlamydospores. These constitute the inoculum.

11. Their color (lighter on one side), markings and variations in form. (See demonstration microscope.) DRAW five different spores.

While the chlamydospores of many smut fungi are resting spores those of *U. tritici*, on account of the blossom infecting habit of the pathogene, have largely lost their ability to remain viable for a long period.

The dusty dry chlamydospores are scattered by the wind over the blossoming wheat. Some spores fall upon the exposed stigmas protruding from the open glumes of healthy heads. Here they germinate, sending a long germtube along and into the stigmatic filaments and by way of them into the young ovary. Germinate spores in water to which a bit of sugar has been added, study and OBSERVE:—

12. The long germtubes, septate and with knee-joint fusions.

13. The uniform absence of sporidia.

DRAW several germinating chlamydospores to show variation.

This germtube is morphologically a basidium which in most other species of Ustilago produces basidiospores (sporidia). In this species, sporidial production is unnecessary. After penetrating to the ovary, the germtube branches and, establishing itself near the growing point, goes into a dormant condition along with the ripening kernel. The invaded seed shows no evidence of injury.

As the embryonic plant develops during germination, the mycelium, aroused by the same condition of heat and moisture (and probably by the activities of the embryo as well), begins active growth, branches and spreads into the stooling culms, and forces its way upward through the tissue as the culm lengthens. When the wheat heads begin to form, the mycelium of the fungus takes possession, completely replacing the more tender or succulent tissues of the spikelet with its dense mats of profusely branching mycelium, finally forming the black, easily ruptured sori of the smut. The diseased heads emerge from the sheath about the time the flowers on the healthy heads are in full bloom. Why?

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LOOSE SMUT OF OATS

There are two smuts of oats, the loose and the covered. Loose smut is generally the more common and the one usually referred to in speaking of oat smut. This disease occurs wherever oats are grown and may often destroy from 25 to 50 per cent. of the crop. Its control with formaldehyde is exceedingly simple and effective.

SYMPTOMATOLOGY

The signs of disease predominate in the symptomatology of the oat smut as they do in most smut diseases of cereals and grasses. Certain morphologic symptoms are also exhibited by the diseased plants.

Signs. Compare the panicles of diseased and healthy plants. Observe:—

1. That the spikelets of the diseased inflorescence consist largely of the black powdery spore mass of the pathogene.

2. That on removing the spore mass from a spikelet by tapping and blowing, there remains shred-like bits of suscept tissue upon which the spores were supported.

3. That occasionally sori are produced in the blade of the uppermost leaf, forming long parallel black striations. (See illustration specimens.)

Morphologic Symptoms. The symptoms of this disease are best studied in the field. One will find:—

4. A distinct dwarfing of the plants, at flowering time when the smutted heads are fully exerted. Diseased culms are decidedly shorter than healthy ones, due chiefly to a failure of the upper internode to lengthen fully.

5. A shortening of the pedicels of the spikelets, giving the smutted panicles a compact erect habit in marked contrast to the spreading panicles of healthy plants. This is evident in the dried specimens before you.

6. That usually all the culms in a stool are smutted, though sometimes one or more may produce healthy panicles.

7. That not infrequently the spikelets of the upper part of the panicle escape invasion by the pathogene and develop normally.

The effects of the disease on the organs of the spikelet may best be studied by carefully dissecting and comparing a diseased and a healthy spikelet (after soaking a while in water). The dissection is best made in water in a watch glass. Observe:—

8. The suppression of awns and the abortion of the glumes, stamens and pistils to mere remnants or shreds of vascular tissues.

9. That in some cases only the base of the glumes are shredded.

10. That the glume tissues which are present in diseased spikelets are pale and blanched in contrast to the green color of these in the healthy spikelets.

Make drawings to show (*a*) the contrast between a diseased and a healthy panicle. (*b*) The leaf lesions. (*c*) The parts of the dissected diseased and healthy spikelets.

Compare specimens of loose smut with those of covered smut. SKETCH to show differences. (See Stevens and Hall 1921, fig. 156–157.)

ETIOLOGY

The pathogene causing the loose smut of oats is *Ustilago avenæ* (Persoon) Jensen. It is closely related to *Ustilago tritici* (Pers.) Jens, which causes loose smut of wheat. The covered smut pathogene, *Ustilago levis* (Kell. and Sw.) Mag., occurs along with *U. avenæ* from which it is most easily distinguished by its smooth, granular spores.

Life History. The life history of this pathogene may be regarded as consisting of primary cycles only. While not so intimately associated with the living suscept during the resting period as is *Ustilago tritici* (Pers.) Jens., it normally exhibits no saprogenic activity under natural conditions.

Primary Cycles are initiated with inoculation of the flowers at blossoming time.

Pathogenesis. The smutted plants scattered through the field are the sources of inoculum.

Remove a bit of the black mass from a smutted head to a drop of KOH solution on a slide; cover and examine. OBSERVE:—

11. The numerous brown, globose bodies scattered through the mount,—the chlamydospores.

12. Their color (lighter on one side); markings and variations in size. DRAW three different spores. (See demonstration micrscope, oil-immersion).

These chlamydospores may retain their vitality for several years if kept quite dry in the laboratory.

These chlamydospores falling within the open oat blossom lodge for the most part upon the sticky feathery stigma. Here according to Zade (1922) they germinate at once producing their characteristic promycelium with sporidia. These structures are easily observed.

Sow some spores on the surface of drops of water (to which a bit of sugar has been added) on slides and incubate for a day or so. Examine under the microscope and OBSERVE:—

13. The rather long hyaline germtube or promycelium,—the basidium of this fungus.

14. That it is septate; number of cells into which it is divided; the crack in the chlamydospore wall where the promycelium emerges.

15. That from each cell at the septum (above or below?) arises one or more oblong sporidia,—the basidiospores.

16. The variations from the normal germination.

Make DRAWINGS to show germination of chlamydospores and formation of sporidia, with such variations as are observed.

17. Examine the mount of germinated spores to see if you can find any of the sporidia germinating. DRAW.

The sporidia borne on the promycelium from the chlamydospore lodged on the stigma, come in contact with the inside of the young oat hull. Here they germinate forming a mycelium which penetrates and spreads through the inner wall tissue of the hull. As the grain ripens and the hull tissues die this mycelium becomes dormant. The mycelium from the sporidia appears never to invade the young growing ovary (?), which now a mature kernel, lies uninfected within a hull whose inner walls are grown through and through with the dormant mycelium of the pathogene.

When the grain is sown in the soil in the spring this dormant mycelium becomes active again, sending forth infection hyphæ which penetrate the mesocotyl of the sprout as it emerges from the seed coat. These hyphæ branch and penetrate between the cells, spreading upward into the growing point. As the shoot grows and branches to form the stool the mycelium keeping pace with the growing tips, spreads into and upward with the new shoots. Usually every culm in the stool is infested. The mycelium at first stimulates the growth of the plant. The activities of the mycelium continue throughout the growth and development of the oat until the formation of the flowers begins. In these rapidly developing embryonic tissues, the mycelium takes possession, appropriates the abundant food substances and begins the formation of chlamydospores.

The formation of these chlamydospores takes place as follows:—The mycelium in the flower at the time that spore formation begins shows a nodulate appearance and the branches are closely fasicled like clusters of grapes. Within each swollen part of the mycelium a chlamydospore is formed. As the chlamydospores mature, the encompassing walls of the parent hyphæ and much of the general mycelium which is not differentiated into spores, gelatinizes or otherwise breaks up and the spores are set free in large masses.

Practically all the floral parts of every spikelet are displaced by the black spore mass of the pathogene. The smutted panicles emerge, the spore masses ripen, and the spores are scattered when the healthy panicles are in full bloom.

Note:—The old theories (1) that the chlamydospores lodge on the exterior of the hulls, remain there until the grain is sown and infect the seedling as it emerges from the hull and (2) that the chlamydospore lodges within the open hulls at blossom time, but does not germinate until after the grain matures and is sown in the soil the following spring; will I believe both have to be abandoned in the light of Zade's work.

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STINKING SMUT OF WHEAT

Although the loose smut is the more common on wheat in eastern United States, the stinking smut is frequently serious. In the great wheat fields of the west it is the common and destructive wheat smut. The losses from the stinking smut may be large not only from the reduction in yield but also from the lowering of the market value of grain with which the smutted kernels become mixed. The life history of the causal fungus is very different from that inducing the loose smut of wheat.

SYMPTOMATOLOGY

The symptomatology of stinking smut varies rather markedly from that of other cereal smuts in that here the signs of the disease are a much less prominent feature of the symptom complex in the growing plants.

The **Morphologic Symptoms** of this disease have been fully described by Barrus (1916) and his observations may be largely confirmed by a comparative study of the diseased and healthy wheat heads provided. These were collected just before flowering time and preserved in formalin. Remove a spikelet from each head, dissect in water in a watch glass and OBSERVE:—

1. That the ovary in the diseased flower is larger, and its pistil more fully developed than that of the healthy flower; it is greenish instead of white.
2. That the diseased stamens are dwarfed, small and yellowish in striking contrast to the plump greenish stamens of the healthy flower.
3. That as the flowers develop the diseased kernels become ovate, pointed, while the healthy ones become waisted and flat topped.
4. The relative size, shape, color and weight of the mature diseased kernel ("spore ball") compared with a ripe, healthy grain. Test them as to comparative specific gravity. Measure several of each as to length and diameter.

Make a series of DRAWINGS to show the comparative morphologic development of diseased and healthy kernels.

Just after the emergence of the ears from the upper leaf sheath certain striking differences between diseased and healthy heads become evident. These are best studied in the field. THEY ARE:—

5. Diseased heads of a bluish-green color; healthy, yellowish green.
6. Diseased heads slimmer than healthy; fœtid odor can now be detected.

At flowering time further differences become apparent:—

7. Diseased plants dwarfed, heads shorter and slimmer, stamens not exserted.
8. All culms in the stool usually bearing diseased heads.

As the grain ripens diseased heads remain erect and rigid instead of bending over like the sound ones. Other differences in the appearance of diseased and healthy heads have been pointed out by Barrus. (Study his paper carefully.)

ETIOLOGY

This disease is caused by *Tilletia tritici* (Bjerkander) Winter, or by *Tilletia fœtens* (Berkley and Curtis) Trelease (= *T. lævis* Kühn). This is a case in which two distinctly different species of fungi cause the same type of disease, in fact the two species may occur together in the same ovary. It is the latter form, *T. fœtens*, which is most commonly found in eastern United States and will be the one here considered. These are species of the Ustilaginales, family Tilletiaceæ.

Life History. This pathogene exhibits only the primary cycles in its life history. Since the chlamydospores become attached to the healthy grains during harvesting or threshing and so are almost continuously associated with the living suscept, this smut fungus like most others may be said to have normally no saprogenic phase. Infection from spores in the soil appears to be rare for this species. The sown wheat in the Pacific Coast regions becomes inoculated by wind borne spores of *T. tritici* in the soil.

Pathogenesis. Inoculation, as indicated above, occurs at threshing time, when spores from the crushed smutted kernels become attached to the healthy grains. Crush one of the diseased grains between thumb and finger. NOTE:—

9. The greasy character of the spore mass. This evidently helps to attach the spores to the seed coat. Note the odor.

Mount a bit of the spore mass in a drop of water. Cover and examine. OBSERVE:—

10. The numerous, more or less globose, smooth brown spores,—the chlamydospores. DRAW.

Compare with these, chlamydospores of *Tilletia tritici* (under the demonstration microscope). Note the reticulated surface of the spores of the latter, the globose shape and smaller size. DRAW.

Make a mount containing chlamydospores of both *Ustilago tritici* and *Tilletia fœtens*. Compare as to size, shape, color and markings. DRAW to show the contrast.

When the inoculated grains are planted, the spores of the pathogene are planted with them. The conditions which favor seed germination also favor spore germination. A promycelium is formed bearing sporidia which in turn produce germtubes capable of penetrating the seedling, but only at some point below the first node or tillering point. Study the germinated spores or the figures in Washington Bul. 126, fig. 2, and OBSERVE:—

11. The germtube arising from the chlamydospores,—the promycelium.

12. The cluster of sickel-shaped sporidia at the end of the promycelium; some bearing secondary sporidia.

13. That frequently two of the sporidia have joined by a short tube forming an H-like figure. This is very common and was once supposed to be a sexual process but it is not now so considered.

Make DRAWINGS to show chlamydospore germination and formation of secondary sporidia.

The germtubes from sporidia or secondary sporidia penetrate the seedling as it emerges from the grain, and branching, form a mycelium. This mycelium quickly reaches the growing point and grows upward within the culm causing no apparent injury to the plant. At the time of flower formation, however, the hyphæ develop rapidly and growing into the ovary replace its tissues with the black spore mass.

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STEM RUST OF CEREALS

Of the many rust diseases of cereals, the black stem rust is perhaps of greatest economic importance. It affects wheat, oats, barley, rye and many other grasses. Young barberry leaves are also subject to the disease. It is one of the earliest recorded rust diseases and the subject of many researches by mycologists and pathologists.

SYMPTOMATOLOGY

The most striking features of the symptomatology of this disease are the fruit bodies of the pathogene. Since these are characteristic of distinct stages in the life of the rust fungus, **Signs** and **Symptoms** may best be studied together in succeeding stages.

Cluster cup stage. The leaves and flower racemes of the barberry are the organs affected. Examine the leaves provided and OBSERVE:—

1. The small lesions; size, location, color both above and below, and the hyperplastic thickening of the leaf tissues in the lesion. The disease exhibits distinct hyperplastic effects on this suscept.

2. With the hand lens, the tiny dark amber or nearly black pimples embedded in the tissues of the upper surface,—the pycnidia (spermagonia), functionless structures of the pathogene. They normally accompany or precede, in the same lesion, the æcial fruit bodies next to be observed.

3. On the lower surface, the sunken cups, exposing their yellow spore masses. These are the æcia or cluster cups.

Make DRAWINGS to show the cluster cup symptoms on both surfaces of the leaf.

Examine the diseased flower clusters. OBSERVE:—

4. The swollen and malformed flowers and pedicels; color of the affected organs.

5. The abundantly developed æcia. Are pycnia present?

SKETCH a diseased flower cluster.

Red rust stage. Examine the specimens of diseased wheat or oats provided and OBSERVE:—

6. The large reddish yellow pustules scattered over the leaf sheath,—the uredinia; not usually so common nor so numerous on the blades. On what other organs do they occur?

7. The size, arrangement and relation of these pustules to the sheaths and leaves. Note the torn edges of the epidermal covering and the dusty spore mass of the ruptured uredinium; its color.

8. The effect on the tissues.

SKETCH a rusted leaf sheath to show the symptoms of the red rust stage. Make a DRAWING of one of the uredinia as it appears under the hand lens.

Black rust stage. This develops during the latter part of the growing season along with the red rust stage so that the two are often observed together on the leaf sheaths and stems. In the material provided, OBSERVE:—

9. The large black linear sori extending up and down the stem,—the telia.

10. The thin epidermal covering, ruptured in most of the telia but partially or wholly covering others; the black spore mass within. Is it also pulverulent like the uredinial spore masses?

11. The telia developed on the inflorescence. What organs are affected?

12. Kernels from badly diseased plants in comparison with those from healthy plants. How do they appear to be affected? Telial sori are occasionally developed under the seed coat of the kernel. (See Phytopath. **1** : 150–154, fig. 1–2).

SKETCH an internode and also parts of the inflorescence to show the black rust symptoms. Make an enlarged DRAWING of a telium as seen under the hand lens.

The **Histologic Symptoms** of this disease may be studied in fresh or alcoholic material of the lesions on barberry or in fresh uredinial lesions on the leaves or stems of wheat or oats.

Make thin cross-section through such lesions and determine (under the microscope) what changes in the tissues have resulted from the presence of the pathogene (Allen 1923 : 601–602, pl. 1–7).

Make detailed DRAWINGS to show histologic modifications.

ETIOLOGY

The stem rust of cereals and grasses is caused by the fungus *Puccinia graminis* Persoon. It is one of the Uredinales which are perhaps the most highly specialized of basidiomycetous pathogenes. It is a heterœcious parasite producing its æcial structures on the barberry, a host widely remote in relationship from its grass hosts on which the other spore forms appear. This species, *Puccinia graminis*, includes a variety of biologic forms, morphologically alike, and all having their æcia on the barberry but restricted rather sharply in their uredinial and telial stages to definite cereals or grasses (Stakmand and Piemeisel 1917 : 491–493).

Life History. This pathogene produces, during its life history, all the known fruiting structures of the rust fungi: pycnia (0), æcia (I), uredinia (II), telia (III), and basidia (IV), each with their characteristic spores. It is therefore said to be a Eupuccinia, that is, a true Puccinia (McAlpine 1906 : 10–11).

The **Primary Cycles** are normally those which develop upon the barberry producing only the pycnia and æcia. It seems certain, however, that the fungus may winter over in the uredinial form (probably as mycelium) and produce uredospores the following spring which initiate primary infections on grasses and cereals. This second type of primary cycle is essentially secondary in all respects except as to the time it originates. It will not be considered separately in this exercise.

Pathogenesis. The inoculum for the primary infections consists of the basidiospores (sporidia), produced on the basidium (promycelium) from the overwintered teliospores on the straw. Germinate teliospores on slides or study the illustrations provided and OBSERVE:—

13. The long slender basidium put forth from each cell of the teliospore; four-celled near the apex.

14. The long pointed sterigma from each basidial cell bearing at its apex a thin-walled ovoid sporidium (basidiospore). Each sporidium contains one nucleus and finely granular protoplasm.

DRAW OR COPY to show sporidia ready for dissemination.

These sporidia are carried by the wind or splashing rain to nearby barberry bushes where in the moisture on the surface, they send forth a germtube which penetrates the epidermis and gives rise to the locally spreading mycelium. From the mycelium, within less than a month, are produced first pycnia and promptly after them, æcia.

Study cross-sections of the leaf (freehand or prepared) through an æcial lesion. OBSERVE:—

15. The large cup-like æcia and opposite them on the upper surface of the leaf, the minute flask-shaped pycnia (spermagonia).

Locate a pycnium and study its structure under high power. MAKE OUT:—

16. The very slender interwoven mycelium forming the pycnial wall.

17. The long slender sporophores arising from the wall mycelium and converging at the center of the pycnium about a small cavity which is filled with very minute bacterium-like pycnospores (spermatia).

18. The protruding neck of the ostiolum through which the spermatia are discharged. These spermatia have now no known function. They will not germinate and are supposed to be vestigial male cells (sperms).

Study an æcium and OBSERVE:—

19. That it is sunken in the tissues; at first covered by the epidermis of the leaf (see one that has not burst through). The pycnia are more superficial; subepidermal or subcuticular?

20. The fine interwoven mycelial threads between the leaf cells about the æcia. Do they ever penetrate into the cells of the suscept?

21. That it is from a densely interwoven mat of this mycelium (to be seen at the base) that the structures of the æcium are developed.

22. The closely packed club-shaped sporophores arising from the mycelial mat in the base of the cup.

23. The chains of spores cut off from each sporophore, forming the parenchyma-like mass that fills the cup. Why are the æciospores here angular in outline? Note that the maturing spores at the mouth of the æcium become globose.

24. The large cells lining the cup. They also arise in chains from short mycelial branches similar to sporophores. Note the difference in thickness of the wall of the inner and outer faces of the cells. This lining of the cup is called the peridium.

The mycelium of the æcial stage arises from the uninucleate sporidium and each mycelial cell is uninucleate. When the formation of the æcium begins, there is a sexual fusion of many pairs of mycelial tips in the region where the base of the cup is to be. From each pair of branches arises a single binucleate stalk,—a sporophore of the æcium. A chain of spores is developed from each of these binucleate sporophores by successive conjugate divisions of the paired nuclei and the laying down of a septum between the resulting pairs thus formed. Examine stained sections carefully under high power and LOCATE:—

25. The paired nuclei in the sporophores; in the æciospores.

Make a DRAWING to show the structure of the æcium, pycnium and their relation to the leaf tissues as seen in section.

Remove with the needle or scalpel some mature æciospores from the cups on diseased leaves provided. If dry, mount in KOH solution. Study and OBSERVE:—

26. Their form, size, color and contents; markings on the epispore. DRAW several æciospores.

But one crop of æciospores is produced. With the maturity and discharge of the last spore, the activities of the primary cycles cease. The æciospores initiate secondary cycles on the cereals or grasses through which the pathogene perpetuates itself until the following year. There is no saprogenesis in the primary cycles.

Secondary Cycles, especially the late ones, develop chiefly on the leaf sheaths and stems of the wheat, oats or grass suscepts. Leaves may also become infected.

Pathogenesis. The first secondary cycles are initiated by æciospores which, falling upon the suscept, germinate in the dew or raindrops sending out a germtube which grows along over the surface until its tip reaches a stoma through which it enters into the tissues within. The protoplasm of the spore passes out into the germtube keeping constantly in its tip as it grows. Within the substomatal cavity, the tip of the germtube applies itself to one of the parenchyma cells and sends into it a short blunt branch,—a haustorium.

Having thus established a food relation with the suscept, a binucleate ramifying mycelium is developed. This mycelium produces mature uredinia within two or three weeks.

Study cross-sections (free-hand or prepared) of a stem through a uredinium and MAKE OUT:—

27. The structure of the sorus and its relation to the tissues.

28. The intercellular mycelium. Can haustoria be found?

29. The stout sporophores arising from the mycelium, each bearing at its apex a uredospore in some stage of development. How can one tell whether the spore is young or mature?

Make a DRAWING of a cross-section through the uredinium.

Scrape some uredospores from a diseased stalk; mount (in KOH if from dried material) and OBSERVE:—

30. The form, size, color and contents of the uredospore as compared with the æciospores; markings on the epispore, if any.

These uredospores are usually wind disseminated, initiating other secondary cycles. They germinate at once in moisture, even in a moist atmosphere. They infect the suscept in a manner and under conditions similar to the æciospores. The germtubes emerge through special pores or thin places in the spore wall. Mount and examine uredospores in lactic acid (heated). OBSERVE:—

31. The size, number and location of the pores. DRAW.

For germination and entrance into the suscept study illustrations provided. Supplement this with a study of spores germinated on slides. Make DRAWINGS to show the points brought out.

From the mycelium produced from the uredospore, other uredinia are produced. This may be repeated seven or eight times during the growing season. The mycelium remains uninucleate throughout all these cycles.

As the suscept begins to mature, there is developed from the mycelium in the secondary lesions, not uredospores but another spore form,—chlamydospores, black resting spores known as teliospores (teleutospores). They often arise at first in the same sorus with the uresospores, later they appear in sori in which no uredospores are formed. Mount some teliospores and OBSERVE:—

32. Their form, size and structure as compared with uredospores; color, where located, in walls or contents?

33. The thick walls and densely granular oily contents.

34. The light spot in the center of each cell; not always evident. The nuclei are located here. These spores are at first binucleate but at, or just before germination, the nuclei fuse and sexual fertilization, begun by the association of nuclei in the æcium, is now completed.

DRAW several teliospores to show structure and variations in form and size observed.

Saprogenesis. With the ripening or death of the suscept, teliospores have been formed usually in great abundance on the straw. When the grain is harvested, many teliospores are left in the stubble. Those in the straw go with it into the stack, then into the manure pile, and finally with the manure back on the land. Under the conditions of moisture and temperature that starts the barberry into growth in the spring, the teliospores on the old straw become active. They germinate *in situ* forming a promycelium from each cell of the spore. Four basidiospores are produced from each basidium. Each is uninucleate, its nucleus having been derived through the reduction division of the fusion nucleus of the teliospore. These sporidia, as we have seen, constitute the primary inoculum which infects the young leaves of the barberry. They cannot, so far as is known, infect any of the cereals or grasses.

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CARNATION RUST

THIS is a disease of European origin, which was first observed in this country in 1890. It rapidly developed in epiphytotic form, shortly appearing in nearly every carnation house throughout eastern United States. It was for a decade the most destructive and most feared of carnation diseases, but is now enphytotic and rarely serious, due to the development of resistant varieties. It also affects some other species of the genus Dianthus and some species in related genera. In Europe the cluster cup stage of this rust is known, affecting *Eurphorbia gerardiana* Jacq.

SYMPTOMATOLOGY

Since the cluster cup stage of the carnation rust is unknown in America, the disease will be studied only as it appears on the common greenhouse carnation.

Signs and **Symptoms** will be considered together since the red and black rust stages appear together and are much alike. Both leaves and stems are affected. Study the materials provided and OBSERVE:—

1. The pale green or yellowish areas on the leaves; which surface?
2. In some of these spots, dark brown or black pustules,—sori of the pathogene. The lighter colored ones are uredinia, the darker ones, telia.
3. The arrangement of these sori within the lesion; scattered, confluent, linear?
4. Some of the sori bursted open exposing the dark colored rusty spore mass within, the ruptured epidermis forming a papery fringe.
5. The sori on the stems; usually darker than those on the leaf. Stem sori are largely telia.
6. The general effect of the disease on badly rusted plants.

Make DRAWINGS to show the characters of the lesions on leaves and stems.

To study the **Histologic Symptoms** make thin cross-sections through leaf lesions. Comparing the cells in the lesion with those of adjacent healthy tissues, determine what changes are induced by the pathogene.

Make detailed DRAWINGS to show histologic symptoms.

ETIOLOGY

The causal organism is a urediniaceous fungus, *Uromyces caryophyllinus* (Shrank) Winter. It is heterœcious, having all the spore forms characteristic of the rust fungi.

Life History. Being, in this country, a pathogene of a greenhouse crop, the fungus is able to propagate itself continuously through secondary cycles. The primary cycle therefore will not be here considered.

The **Secondary Cycles** are started by uredospores from sori on leaves or stems of living plants (except of course when æciospores occur). They are constantly being initiated as uredospores are matured and disseminated.

Scrape some of the uredospores from a sorus; mount and study, OBSERVE:—

7. The form, size, color, thickness of wall and markings on the epipspore. Heat some in lactic acid and see if the number and arrangement of the germpores can be determined. DRAW several uredospores.

The uredospores are scattered by wind or splashed, in watering, to nearby healthy leaves and plants. They germinate very readily in water. Germinate some of them. OBSERVE:—

8. The number of germtubes produced from each spore. Are they septate?

9. The densely granular protoplasm crowded into the tips of the long germtubes. The empty germinated spores. Compare with those not germinated.

DRAW two or more germinated uredospores.

The germtube enters through a stroma and forces its tip between the palisade cells, sending haustoria into them, thus establishing a food relation with the suscept. Make thin cross-sections of a leaf through a sorus; clear in chloral hydrate; study and OBSERVE:—

10. The intercellular mycelium; often in gnarled and interwoven masses, especially in the intercellular spaces, prying apart and isolating the leaf cells; densely granular, septate.

11. The peculiar lobed haustoria in cells adjacent to the mycelium; their very thin walls and densely granular protoplasm.

12. The uredinium; its structure and relation to the leaf tissues.

13. The matted mycelial bed from which arise the short closely crowded uredosporophores.

14. The globose uredospores; thin walled, covered with short spines and much lighter colored than the one-celled teliospores, some of which are usually to be found in a uredinium.

Make a DRAWING of a portion of the cross-section through a sorus to show the pathogene structures and their relation to the leaf tissues.

The mycelium is supposed to spread very generally throughout the leaf and stem tissues beyond the point where distinct lesions appear, so that cuttings from diseased plants are usually infected. It is believed that this habit of the pathogene is responsible for its wide and rapid distribution in this country. Make thin sections through the leaf at some distance from the lesion; clear in chloral hydrate; examine and DETERMINE:—

15. Whether or not mycelial invasion is general.

Select a dark colored sorus,—a telium (usually more abundant on stems); mount some of the teliospores. Study and OBSERVE:—

16. That they are much darker in color than the uredospores, although of about the same size and shape; walls slightly thicker, and epispore nearly or quite smooth; stalk often still attached. DRAW several.

The teliospores arise from the same mycelium on which uredospores are produced; usually more abundant in the sori formed as the affected organs grow older.

Saprogenesis. The teliospores do not germinate at once as do the uredospores, but require a period of rest. They are chlamydospores, and, under natural conditions, germinate in the spring producing a promycelium with four sterigmata, each bearing a basidiospore (sporidium). Study the drawings provided. COPY.

These sporidia produced by the germination of the teliospores can, so far as known, infect only the Euphorbia suscept. As *Euphorbia gerardiana* does not grow in this country, the sporidia cannot function in the propagation of the pathogene, and primary cycles cannot develop.

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SNAPDRAGON RUST

THE rust of snapdragons which was discovered in California in 1895 is now common throughout northern United States. It also occurs in Bermuda. It is destructive both in the greenhouse and the garden.

SYMPTOMATOLOGY

The symptoms of this disease may occur on the snapdragon at any age from seedling to blooming plant. They usually appear however about the time the plants are a third to half grown. Leaf blades, petioles, stems, calyces, and occasionally the seed pods may show the lesions.

Certain **Morphologic Symptoms** precede the appearance of the characteristic signs of the disease. In specimens showing the early stages of the disease OBSERVE:—

1. The slightly raised yellow patches on the under surface of the leaves; appearance of the lesion on the upper surface.
2. A slight curling of some of the affected leaves.

Examine diseased plants in the final stages of the malady. OBSERVE:—

3. The small, prematurely opened flowers.
4. The wilted foliage and blackened aspect of the plant. The leaves finally die and shrivel (Peltier 1919, fig. 1).

The **Signs** of the disease develop shortly after the appearance of the yellow patches. In the specimens (or photographs) provided OBSERVE:—

5. The chocolate brown powdery pustules scattered over the surface of the yellow lesions. On which side of the leaf?
6. Their form, size and arrangement (Peltier 1919, fig. 2). These are the uredinial sori of the pathogene.
7. The relative size, shape, and distribution of these sori on the stems and other organs of the suscept.
8. The ruptured epidermis about the sori.
9. The dusty spore mass within; color in mass on white paper.
10. The black telial sori; most commonly to be found on the stems.
11. Their form, size and relative abundance as compared with the uredinia.
12. That the teliospore mass is not powdery like that of the uredospores.

Make DRAWINGS to show these signs and symptoms.

The **Histologic Symptoms** are best observed in free-hand sections through young leaf lesions. Study such sections and OBSERVE:—

13. Which tissues are affected. Are the cells of the upper epidermis and palisade layers modified?
14. Effect on the size and shape of the cells of the spongy parenchyma in the immediate vicinity of the sorus.
15. The wedging effect of the mycelial strands and masses.

16. The nature of the injury to the cells of the lower epidermis where the sorus bursts through.

17. Effect of the pathogene on the pink pigment of those cells where it normally occurs.

18. Effect on the color and form of the chloroplasts.

Make a detailed DRAWING of enough of the section to show the histologic modifications.

ETIOLOGY

Snapdragon rust is caused by the uredinaceous pathogene, *Puccinia antirrhini* Dietel and Holway. The history of its discovery and spread over its present range is an interesting one (Peltier 1919 : 535–536).

The **Life History** of this pathogene is not fully known (Mains 1924 : 281–287). Although uredospores and teliospores are both formed and the latter have been observed to germinate, no æcial stage is known. Since the fungus is primarily a greenhouse pest, escaping to the garden on cuttings, infected seedlings or as wind borne uredospores no overwintering stage is necessary to its perpetuation on the cultivated snapdragon. In warm regions like California and Bermuda it carries over on old plants left standing through the off season.

Only **Secondary Cycles** may be said to occur, as the primary cycles are as yet unknown.

Pathogenesis. Diseased plants are the sources of inoculum; the inoculum the uredospores. Mount some of the uredospores in a drop of water and under the high power OBSERVE:—

19. Their size, color, shape, markings and contents. DRAW.

Heat some of the uredospores in lactic acid on a slide, cover and EXAMINE:—

20. For the germpores; location, form, size and color. These pores are thin spots in the epispore from which the germtubes arise.

DRAW to show germpores.

To germinate uredospores remove some of the powdery brown mass from a sorus and mount in a drop of fresh tap water on a slide. Place in a petri dish moist chamber and set them in the ice box for five to eight hours. These spores germinate best at about 10° C. Examine the germinated spores and OBSERVE:—

21. Number, length, color, septation and contents of the germtubes from each spore. What percentage has germinated? (Doran 1921 : 43–46.)

DRAW several germinated uredospores.

The germtubes enter through stomata on the upper surface of the leaf (Doran, 1921 : 51–53). The resulting mycelium spreads locally in the tissues causing the lesions already observed. Cut thin sections through a young lesion in which the sori are just bursting through. Stain with eosin. OBSERVE:—

22. The mycelium; color, septation and relation to the cells of the leaf. In what tissues is it most abundant?

23. The haustoria in cells adjacent to the mycelium; their form and number per cell.

24. The stroma-like mat of mycelium about the base of the sorus.

25. The sorus; form and relation to the leaf tissues; the ruptured epidermis, upper or lower?

26. The short crowded sporophores bearing uredospores.

Make a detailed DRAWING of a part of the section to show the pathogene structures observed and their relation to the leaf tissues.

The ripe uredospores scattered by splashing water or wind initiate new secondary cycles. As the plants grow old or slowly dry out telia are formed, most abundantly on the stems. Cut thin sections through a telium on the stem. Study under the microscope and OBSERVE:—

27. The structures of the telium.

28. The form, size and color of the teliospores. Note that they are two celled and smooth.

DRAW to show structure of telium and teliospores.

Although earlier investigators failed to obtain germination of the teliospores they have recently been germinated by Hockey and by Mains. The conditions governing their germination are not understood and their role in the life history of the pathogene is of doubtful importance. Mains (1924) believes the fungus to be heterœcious, producing æcia on some other plant.

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DODDER

The term dodder is applied as a common name to the pathogenes as well as to the diseases which they cause. It is used for all the diseases caused by species of phanerogamous parasites of the genus Cuscuta. The different dodder diseases are often distinguished by adding the name of the suscept, as alfalfa dodder, clover dodder and the like. The pathologic effects are in all cases similar.

SYMPTOMATOLOGY

All parts of the suscept above ground, especially the stems, are directly attacked by the dodder. The most striking evidence of the disease is the dodder itself.

Signs. The tangled yellow stems matted about the suscept form conspicuous yellow patches in the field. These doddered areas, often several feet in diameter, are at first more or less circular. Examine the photographs showing spots in the field. OBSERVE:—

1. The dwarfed and dying plants at the center of the doddered area.
2. The densely matted dodder stems.
3. The long spreading runner-like dodder stems about the margins of the area, extending out among the healthy plants.

Examine the specimens provided and OBSERVE:—

4. The yellow dodder vine entwined about the stems of the suscept; size, color shape in cross-section, branching; the clusters of flowers.

Make a SKETCH to show the general aspect of the dodder on the suscept.

The **Morphologic Symptoms** of the disease are not usually striking, but the effects on diseased plants as far as they are to be detected are hypoplastic in character. Compare diseased and healthy plants. OBSERVE:—

5. Dwarfing of the diseased plant as a whole and yellowing of the foliage. Badly attacked plants are eventually killed.

The **Histologic Symptoms** are not marked as the pathogene causes little injury to the tissues at the points where it attaches itself to the suscept.

ETIOLOGY

Dodder diseases are caused by species of Cuscuta, a genus of the family Convolvulaceæ. Some near relatives in the same family are the morning glories, bindweeds, buckwheat and sweet potatoes. There are about ninety species of Cuscuta, of which about half occur in America. The most common one on cultivated crops like clover and alfalfa is *Cuscuta epithymum* Murray, a species introduced from Europe.

Life History. This pathogene has an annual life history. It is known, however, that it commonly overwinters in a vegetative condition on perennial suscepts like clover and alfalfa (Stewart, et al. 1908 : 369–374).

The **Primary Cycles** are initiated by the seeds which have lain on the ground over winter.

Pathogenesis. Examine seed of *C. epithymum.* NOTE:—

6. Their surface markings; color; and that the one side is convex while the other is flattened in two or more places. Measure several.

DRAW:—(*a*) several seeds to show variations in size and form; (*b*) two seeds greatly enlarged showing all their characteristics. Describe the color in the notes on the margin of the paper.

7. The seed of the other species provided. These may all occur on clover or alfalfa. Make enlarged DRAWINGS of each so that they will be comparable in size with those of the drawings of *C. epithymum.* Examine the photographs of plates taken from Koch's work. OBSERVE:—

8. The embryo coiled in the seed and embedded in the food substance. DRAW.

If available, examine growing dodder seedlings and NOTE:—

9. How they first attack the plant. (See Stewart et al. 1908, pl. 3, and Koch's pl. 1, fig. 1–3.) Make DRAWINGS to show the characters of the seedling and its method of attacking the suscept.

As soon as the seedling has attached itself to the suscept, the vestigial root by which it was anchored in the soil, shrivels and dies. The twining stem, however, feeds on the suscept, grows rapidly and by branching spreads to neighboring plants. Study the dodder plant provided. OBSERVE:—

10. The type of branching; opposite or alternate?

11. The leaves; their location, size, shape, color and abundance. How are these foliage conditions to be accounted for?

Untwine and separate a portion of the dodder thread from the stem of the suscept. OBSERVE:—

12. That it is attached only at certain points. At such places the spirals are shorter and are tight about the stem.

13. With the hand lens, the rows of minute punctures in the stem where the dodder was attached; the oval depression about each.

14. The corresponding row of concave sucker-like disks on the dodder stem,—the attachment disks; the minute elevation at the center of each disk,—the broken-off haustorium.

15. The aborted haustoria in places along the dodder stem where they failed to come in contact with the suscept.

Make a DRAWING of a portion of the suscept with entwined dodder to show all the points brought out in 10–14.

Cut thin cross-sections through the suscept and dodder stems at the point where haustoria are attached. Cover and examine under low power. OBSERVE:—

16. In the suscept stem, the large pith, the vascular cylinder, the cortex, and the epidermal layers. In the vascular cylinder, note the xylem containing the large ducts, and the phlœm which consists of soft tissues within and the lighter area of sclerenchyma fibers without.

17. The haustorium of the dodder. Note the shape of the attachment

disk in section; its relation to the haustorium; where the haustorium arises and how it penetrates the suscept tissues.

18. What happens where the haustorium comes in contact with the sclerenchyma fibers of the suscept? Toward which tissue of the stem does it grow? Why?

19. The spiral ducts in the center of the haustorium.

Make a diagrammatic DRAWING of the section through the stems of the dodder and suscept to show the relative sizes and relation of parts.

With the low power, examine the stained longi-sections of the haustorium. NOTE:—

20. The long thread-like cells extending out from the tip and sides of the haustorium into the cortex, pith, and sometimes into a bundle, making a connection with a conductive vessel of the suscept.

21. The layer of crushed cells at the point where the haustorium emerges from the dodder stem. Does the epidermis of the dodder continue about the haustorium? What becomes of it? Are the cells of the suscept crushed? What is the function of the thread-like cells?

DRAW a single haustorium showing the type of cells in the center and at the ends; the relation of suscept tissues thereto.

Toward autumn flowers may be developed on the dodder vine. Study a fruiting branch of the dodder and OBSERVE:—

22. The compact flower clusters. Where do they seem to be most commonly formed? How many flowers in a cluster?

23. Under the hand lens, the scale-like leaf at the base of each flower cluster and at the base of each flower.

DRAW a single flower cluster enlarged three times.

Dissect and study a single mature flower. NOTE:—

24. The structure and arrangement of the calyx, corolla, stamens with fringed scale at the base of each, and the pistil. How may parts of each? DRAW a single flower enlarged, with calyx and corolla removed at one side.

Cut across the ovary, examine with hand lens and DETERMINE:—

25. How many carpels and ovules are produced. DRAW. Examine the matured capsule. How does it compare in size with the flower? How many seeds does it contain? Note the remnants of the calyx or the corolla at the base. From what does the capsule develop? DRAW.

Saprogenesis. The seeds fall to the ground where they lie dormant until the following spring. They germinate when conditions favorable to growth prevail. The vestigial root serves as an anchorage for the simple slender seedling, probably taking up only water from the soil. The seedling seems to be dependent largely on food stored in the seed, although chlorophyl is slightly developed. Seed germination and contact of the seedling with its suscept completes the primary life cycle.

Secondary Cycles may be initiated during the growing season by pieces of dodder stems broken off in cutting and harvesting the crop. These fall upon healthy plants, infecting them. Pieces of dodder stems are very tenacious of life and thus afford a dangerous inoculum for spreading the pathogene in cultivated crops. The spreading of the pathogene from one individual suscept to a neighboring one may also be regarded as a type of secondary infection.

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HYPERPLASTIC DISEASES

CLUB ROOT

Club root is a common disease of cruciferous crops, especially cabbage, cauliflower and turnips, in temperate regions. It is the best known of the few diseases of plants caused by slime molds.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of the disease are exhibited chiefly in the roots, though there are certain secondary leaf symptoms by which diseased plants may be detected as they stand in the field.

Field symptoms. If available inspect the plants in a cabbage field in which club root is known to occur. Observe:—

1. The dwarfing of diseased plants and failure to head.

2. The flagging or wilting of the leaves, especially at midday. They recover at night.

3. The slightly paler color of affected plants.

4. The swollen and distorted roots revealed on pulling up an affected plant.

Root galls. Comparing the roots of diseased and healthy cabbage plants observe:—

5. The relatively small size and the much more fibrous and branched character of the roots of the healthy plants.

6. The much swollen and enlarged roots of the diseased plants; occasional healthy roots arising from the stems above the lesion. Why? Determine by diameter measurements how many times the diseased roots have been enlarged.

7. That tumefaction occurs either in the region of the root next to the stem or out in the feeding roots. Explain this.

8. That the entire diameter of the root is involved. It is not simply an outgrowth on the side of the root.

9. The specimens of club root on the different suscepts in the illustration material. (See also Vermont Bul. 185, pl. 2–5). Determine whether there is any uniformity in the shape of the swellings which may be regarded as characteristic of this disease on the different suscepts.

10. That the surface of the galls, especially in the old specimens, is much cracked and checked; to what due? These cracks allow the entrance of decay-producing fungi and bacteria. The succulent tissues quickly become soft and rotten and have a foul odor. The club root pathogene itself does not cause the tissues to soften.

11. That the root beyond the gall, the branches and fine feeders, seem to be normal and healthy.

12. The abruptness with which tumefaction ceases at the base of the stem; this is a disease of the roots.

Nodules, tubercules and galls due to other causes are often produced on roots of plants, as for example; the nematode gall caused by minute worms; legume tubercles and crown gall of trees caused by pathogenic bacteria. (See illustration specimens in jars.)

Make a DRAWING of a "clubbed" root along side of a healthy one. Maintain relative proportions.

For the study of **Histologic Symptoms** both free-hand sections and prepared slides should be used. Study a free-hand cross-section through a small healthy root. OBSERVE:—

13. The core of the root, made up of alternating wedges of xylem (vessels and wood cells) and medullary rays. Note that the xylem wedges are frequently fused toward the center of the stele so that the medullary rays are seldom continuous from the cortex to the center.

Stain the section with a solution of iodine and NOTE:—

14. The yellow color taken on by the walls of the lignified wood cells which, with their thicker walls, now contrast more sharply with the medullary ray cells.

15. The bark of the root surrounding the xylem cylinder consisting in large part of the thin-walled cortex cells.

16. The groups of thick-walled sclerenchyma cells in the outer cortex, stained deep yellow by the iodine. What is their function?

17. The ends of the phlœm strands, made up of small thin-walled cells in the inner cortex, each opposite a xylem wedge.

18. The outermost tissues of the root, rough and torn,—the cork layer.

Make a diagrammatic DRAWING 3 inches in diameter showing the anatomy of a healthy cabbage root as seen in cross-section. Make an enlarged and detailed DRAWING of a V-shaped portion of a cross-section showing form and arrangement of cells from center to circumference. Include (in outline) at least one vascular wedge.

Cut, mount, stain with iodine, and study a thin cross-section of a diseased root (or use prepared slide). OBSERVE:—

19. The abnormally increased diameter of the root. To what does this seem to be due; increase in size (hypertrophy) or numbers (hyperplasia) of the cells or both? (Compare with the section of the healthy root.)

20. The xylem vessels in a few small groups. Note their isolation. To what part of the section are they chiefly confined? Why? (Compare Lutman 1913, fig. 3.)

21. The ends of the phlœm strands composed of small regular cells arranged in rows around the outer end of the xylem wedge. The relative amount of phlœm as compared with the amount of xylem; as compared with the amount of phlœm in a bundle in the healthy tissue.

22. The abnormally broad medullary rays, resulting from the increased size of the cells. The dark masses in some of the cells,—the plasmodia or spore masses of the pathogene.

23. The cortex, the most abnormally increased of all the tissues. The

cells are enlarged and perhaps increased in number. Some of them are filled with the brown masses of the pathogene. Most of them show nucleus and cytoplasm. Note the entire absence of sclerenchyma or stone cells; why wanting?

24. The layer of cork cells on the outside. Are these affected?

Make a diagrammatic DRAWING showing the anatomy of the diseased root as seen in cross-section. Make an enlarged and detailed DRAWING of a V-shaped portion of the section from the center to the outside, including one vascular unit and medullary ray. Indicate the presence of the parasite in the cells by fine stipple-work.

ETIOLOGY

This disease is caused by the myxomycete, *Plasmodiophora brassicæ* Woronin. It is apparently an obligate parasite, saprogenesis being chiefly a period of rest. It has never been cultured saprophytically.

Life History. This pathogene has, normally, an annual life history but its spores may probably lie dormant in the soil for several years.

The **Primary Cycle** is initiated by spores that have remained dormant in the soil from a former season.

Pathogenesis. When the galls rot, the spores of the pathogene, mixed with the decayed root tissues, remain in the soil. Selecting a gall in an advanced stage of the disease, remove a bit and crush it in a drop of water on a slide; cover and examine with the high power. OBSERVE:—

25. The minute globose hyaline spores, abundant among the crushed tissue fragments. DRAW.

These spores germinate in the spring after the manner of those of the saprophytic slime molds. The spore wall cracks and the naked protoplast creeps out. Study the process as illustrated by Woronin (Lutman 1913, fig. 6). COPY to show several stages in spore germination and the development of swarmspore or myxameba.

These myxamebæ, thus set free in the soil water, come in contact with nearby roots of susceptible plants and infection results. The single uninucleated swarmspore enters through a root hair (Chupp 1917 : 427–434, fig. 101). Here by rapid nuclear division and growth, a multinucleate ameba or plasmodium is formed which penetrates into the cortical tissues. By direct migration through the cell walls and by the division of the meristematic cells into which the ameba have penetrated, invasion of the various tissues is affected.

The plasmodium increases in size, destroys the protoplast of the cell in which it is lodged, and gradually fills the lumen. Make thin sections (or use prepared slides) through roots in the early stages of the disease. OBSERVE:—

26. The finely and densely granular multinucleate plasmodia of the pathogene in certain of the enlarged cortical or medullary ray cells. Compare with the protoplasm of uninvaded cells nearby. DRAW an uninvaded and a normal cell, with contents, to show contrast.

These multinucleate plasmodia, when mature, divide into spores, each one celled. Cut sections (or use prepared slides) of diseased roots containing mature spores. OBSERVE:—

27. That the infested cells are packed with spores; their uniformity in size; and absence of the capillitium so characteristic of most saprophytic slime molds. The suscept cell serves as a sporganium. DRAW an invaded cell with spores.

In which tissues of the root is the pathogene most abundant?

Saprogenesis. As the galls decay in the autumn, the spores are set free in the soil. Here they are supposed to lie dormant not only during the succeeding winter but many of them are believed to remain for some years in the soil in a viable state. Since the spores germinate readily under favorable conditions of moisture and temperature even in the absence of roots of cruciferous plants, the persistence of the organism in the soil for years is not yet satisfactorily accounted for. There is still the possiblity that it may have an active saprogenic phase in its life cycle.

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LEGUME TUBERCLES

The bacterial nodules which occur on the roots of leguminous plants are not generally regarded as evidence of a diseased condition. They are nevertheless pathologic in character. That the plant usually profits from their presence on its roots is no argument against their pathologic nature. The roots of all our common leguminous crops show these galls.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of this disease should be studied and compared on a number of different legumes. In the specimens provided (including demonstration specimens in jars) OBSERVE:—

1. The shape, size and color of the galls: more or less characteristic for each species of legume.
2. Location on the root system; deep or shallow; small or large roots, lateral or terminal?
3. Types of galls; simple and compound (branched).

Make DRAWINGS to show the types of tubercles on at least four species of legumes.

For a study of **Histologic Symptoms** examine either free-hand or prepared longi-sections of tubercles and normal young root tips. OBSERVE:—

4. The shape and appearance of the longitudinal section of the tubercle. Compare with that of a young lateral root tip.
5. The bacteroidal region. Where located in the gall.
6. The vascular strands, their origin and location with respect to the bacteroidal region.
7. Orientation of the xylem and phlœm in the gall strands compared with their positions in the normal root.
8. What tissue of the root is enlarged to form the bacteroidal region of the gall.
9. Tissue relations at the juncture of the gall and the root from which it arose. Is the cortex of the root continued into the gall? The epidermis?
10. The primordial meristem and root cap. Compare these in the normal root and in the gall.
11. The outer cell layer limiting the bacteroidal region,—the starch sheath.
12. The size and contents of the bacteroidal region of the gall compared with the cells of the same region in the normal root.

Which condition predominates in the gall, hyperplasia or hypertrophy?

Make diagrammatic DRAWINGS of a longi-section of a tubercle and of a normal root tip. Make the drawings large in outline and detail portions of each to show the cellular character of the different tissues. Label fully.

Now study cross-sections of the tubercle. OBSERVE:—

13. The bacteroidal core, ring of vascular bundles, cortex and starch sheath.

Make an outline DRAWING of the cross-section and detail a sector from center to circumference to show cellular structure of the different regions of the gall.

14. The effect of the bacterial invasion on the organs of the invaded cells; nucleus, cytoplasm and cell walls, as compared with those of the homologous cells in a healthy root (Fred 1911 : 123).

Detail in a large DRAWING a healthy and an invaded cell.

ETIOLOGY

The root tubercles of leguminous plants are caused by a bacterial pathogene, *Bacterium leguminosarum* (Frank) E. F. Smith. This species appears to consist of a number of more or less distinct biologic forms.

Life History. The pathogene is a highly specialized parasite, restricted apparently to the roots of legumes, in the growing tissues of which it lives and which it stimulates to excessive growth.

Primary Cycles are probably the only ones which occur. They are initiated in early spring and summer by bacteria left in the soil from decaying tubercles of the previous season.

Pathogenesis. Since the inoculum cannot be readily obtained for study from the soil, mount a bit of the pure culture provided and OBSERVE:—

15. The form and size of the bacteria. They should be actively motile. Each bacterium is equipped in the active stage with a single polar flagellum (to be seen only in especially stained preparations. See demonstration microscope or illustrations). DRAW:—

The active bacteria in the soil accumulate in small groups on the sides of the root hairs. They appear to secrete some substance which softens the walls of the root hair enabling them to penetrate it. The root hair is stimulated to bend or curl about the penetrating bacterial mass. Study the prepared slides (or illustrations) showing root invasion. OBSERVE:—

16. The curled tip and the infection thread extending through the length of the root hair. Of what is this infection thread composed?

17. The infection threads in the cortical cells of the root; the funnel-shaped swellings in the thread at the point where it penetrates the cell wall (Smith 1911, fig. 36). (Best seen under oil immersion, demonstration microscope.)

Make DRAWINGS to show details of root hair penetration and invasion of the root tissues.

The bacteria, having penetrated to meristematic tissue, multiply rapidly in the growing cells and stimulate them to abnormal growth and division; this results in the production of the gall.

Cut thin cross-sections of a tubercle. Examine the bacteroidal region with high power and OBSERVE:—

18. The numerous infection threads extending from cell to cell. This shows how new cells formed from the primordial meristem are invaded from adjoining bacteria-filled cells.

19. The immense number of bacteroids in each invaded cell.

20. That the bacteroids are for the most part not rod-shaped like those in the soil (or culture) but are club-shaped to Y-shaped; most evident in the central cells. As the bacteria in the earliest invaded cells cease to divide they gradually take on these so-called involution forms (Fred 1911 : 134–137).

The degenerate bacteroides are attacked by enzymes of the suscept and the nitrogenous material of which they are largely composed is then made available for the growth and development of the legume itself. Some of the individual bacteria in the outer layers of the bacteroidal tissues remain rod-shaped and are set free in the soil with the disintegration of the gall.

Saprogenesis. Whether during their sojourn in the soil, the bacteria lead an active saprophytic existence, though highly probable, is not certain. They can be readily grown on artificial media. They are not known to produce spores. The disintegrating galls with the dead bacteroides containing a high percentage of nitrogen which was fixed from the free nitrogen of the air, adds greatly to the fertility of the soil for succeeding crops.

References

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PEACH LEAF CURL

This is a common and frequently destructive disease of peaches in nearly every peach growing region. It is especially severe in regions subject to cold, wet weather in the spring. One of our most profitable commercial varieties, the Elberta, is particularly susceptible.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of this disease are of the hyperplastic type. Lesions occur chiefly on the leaves but the current years growth of shoots are frequently deformed. The flowers and fruits are occasionally affected.

Leaf curl. Examine the specimens and illustrations available and OBSERVE:—

1. The flexible character of the normal portions of the leaf. Tear it and note the texture.
2. The wrinkled and distorted portion of the leaf. Tear and compare with the healthy portion as to texture and flexibility. Explain the difference.
3. Relative thickness of diseased and healthy portions of the leaf.
4. Color symptoms exhibited in the lesions. (See colored photographs.) List them.
5. The character of the distortion. In which direction with respect to the long axis is the diseased leaf wrinkled? Why? DRAW.

Examine photographs by Wallace and Whetzel (1910, fig. 83–86 and Pierce 1900, pl. 6 and 7). OBSERVE:—

6. That the diseased leaves soon drop, partially or wholly defoliating the tree.

Twig lesions. Study specimens and illustrations of infected twigs available (Pierce 1900, pl. 1 and 5). OBSERVE:—

7. The stunting of the shoot and resulting rosetting of the leaves due to the failure of the internodes to lengthen.
8. The hypertrophied stem, thickened in linear ridges, sometimes extending down into the previous year's growth along the line of the cambium.
9. The badly infected leaves. The leaves on diseased twigs are more severely affected than on the healthy twigs, for invasion becomes general or systemic and the leaf fundaments become diseased as they are formed. Most of the affected shoots finally die. This is a source of heavy loss in budded nursery stock.

Flower and fruit lesions. If specimens or photographs are available study the symptoms exhibited. DRAW.

Secondary symptoms. We have already noted the most striking secondary symptom,—abscission (defoliation). Severe leaf infection may prevent the fruit from setting or may cause it to drop while still small. (See Pierce 1900, 124, pl. 17 and table 33.)

The only external **Signs** of the disease is a silvery coating of the surface of the leaves due to the layer of mature asci covering it; to be seen only in living specimens.

The **Histologic Symptoms** may be studied best in free-hand sections through lesions on living leaves or in prepared sections. If the latter only are available, select a place in the section where the diseased tissues may be compared with the normal. OBSERVE:—

10. The increased thickness of the leaf in the diseased portion. To what is this due, increase in size, or in number of cells?

11. Which tissues are most affected, palisade or spongy parenchyma?

12. The effect on the number, shape and arrangement of the palisade cells; on the chloroplastids and cell walls. These cells were affected before they had differentiated.

13. The effect on the epidermal cells on the upper surface of the leaf; on the lower surface.

Make a detailed DRAWING showing comparatively the histological structure of the diseased and the normal portions of the leaf. (See Knowles 1887 : 216–218, pl. 13).

ETIOLOGY

The cause of this disease is *Exoascus deformans* (Berkley) Fuckel, one of the primitive ascomycetes of the order Protodiscales and of the family Exoascaceæ. No definite fruit body is formed, the asci developing directly from the mycelium in an exposed hymenium over the surface of the lesion.

Life History. There appears to be but one cycle in the life history of this pathogene,—the primary. There is in nature, apparently only the sexual stage unless one is to consider the budding of the ascospores as a much reduced conidial stage.

The **Primary Cycles** are initiated by the ascospores (budded) the source of inoculum being the diseased leaves.

Pathogenesis. Study cross-sections (free-hand or prepared) of a leaf through a lesion bearing asci. OBSERVE:—

14. The row of enlarged and angular epidermal cells of the leaf; their organs and contents.

15. The layer of long, more or less angular asci, containing spores, standing at right angles on the epidermal cells.

In the prepared slides, OBSERVE:—

16. Some of the asci, small and young with deeply stained protoplasmic contents and nuclei.

17. Mature asci containing deeply stained ascospores or empty; shape and size of the spores; number in an ascus (Pierce 1900, pl. 3).

18. That some of the asci have but eight rather large spores in them while others are packed with many smaller spores, which have resulted from the budding of the large ascospores within the ascus (for method of budding see Pierce 1900, pl. 4).

DRAW an ascus showing the original 8 spores; another filled with ascospore buds.

These bud spores are discharged from the opened asci and scattered. Some fall in the axils of the leaves where they settle on to the forming buds, which thus become the infection courts. At what time of year does this inoculation occur? Examine the buds on the living peach twigs provided. OBSERVE:—

19. The hairy character of the outer bud scales. Remove one and study it under low power. Minute globose spores may sometimes be detected, lodged among the long hairs on the scale. These are the ascospore buds which are passing the winter here during the inactive period of incubation. Make an enlarged diagrammatic SKETCH of an inoculated bud.

The spring rains which cause the buds to swell and to open, also cause the bud spores to germinate and send forth a slender germtube which enters the bud between the scales and penetrates the tender leaves. As the leaves develop, the mycelium within spreads between the cells and stimulates the growing tissue, resulting in a curling and fluting of the blade. The germtube may also penetrate into the growing tissues of the developing twig.

Make thin sections through diseased leaves or twigs; clear in chloral hydrate; wash and stain for some time in eosin or methyl blue; wash and mount in water. LOCATE:—

20. The intercellular mycelium, fitting tightly in, and conforming with the intercellular spaces. No haustoria are formed. DRAW a bit of the mycelium with adjoining suscept cells.

Branches of this mycelium pass toward the surface between the epidermal cells. Make thin tangential sections from the surface of the diseased leaf (or see Pierce 1900, pl. 3). Stain as above and study under the high power. MAKE OUT:—

21. The very abundant subcuticular mycelium; its irregular septate form; its branching and anastomosing habit. From this mycelium arise the asci.

DRAW, showing cross-section of leaf with mycelium and asci.

Saprogenesis. There is so far as known no saprogenic phase. This fungus is an obligate parasite. It may however, be grown in pure culture on a variety of media (Mix. Phytopath. **14** : 217–233, 1924).

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LEAF BLISTER OF OAKS

This is a very common leaf disease of oaks. Frequent epiphytotics, with more or less serious effects on the suscepts occur in southern United States, while in the north very little damage is done. Many species of oaks are affected (Wilcox 1903 : 179–180).

SYMPTOMATOLOGY

The **Morphologic Symptoms** of the disease are very characteristic, the most striking feature being the more or less circular arcuation of areas in the leaf blade, from which the name of the disease is derived. Lesions are recorded on the leaves only.

In the material provided OBSERVE:—

1. The so-called blisters or puffs; their shape and size; number per leaf.

2. That in some cases the spots have become confluent and the entire leaf is much curled.

3. That the blister is usually convex on the upper surface of the leaf and concave on the lower, however, this is not a constant character.

4. That the upper or convex surface of the blister is, in young spots, lighter green than the normal leaf but that in the older spots, it becomes more or less flecked with brown.

5. That the lower or concave surfaces of the blister is a frosty gray color in the fresh condition but changes on drying to a dirty or brownish gray. The coloration and velvety appearance of the under surface is due to the fruiting structures of the causal fungus.

Make a SKETCH to show the symptoms of leaf blister.

According to Wilcox (1903 : 174) defoliation early in the summer frequently occurs in the South, the tree producing a new set of leaves by midsummer by the proleptic development of the buds intended for next year's growth. Repeated defoliation through several years may result in the death of the tree. Defoliation rarely or never occurs in the North.

The **Histologic Symptoms** of the disease are best studied in free-hand sections of fresh leaves. Prepared sections should also be examined.

Study the sections available and OBSERVE:—

6. The hypertrophy in the lower epidermis, spongy mesophyl, palisade layer and upper epidermis.

7. The hyperplasia in the lower epidermis and spongy mesophyl tissues.

8. That in the healthy parts of the leaf the cells of the lower epidermis are longer than broad. The effect of the fungus is to cause these cells to increase greatly in size and, being so tightly packed together, they elongate in a direction perpendicular to the surface of the leaf. Finally septa may be laid down at right angles to their long axes.

9. That the cells of the spongy mesophyl are increased both in number and size and are tightly packed together, largely eliminating the intercellular spaces so common in the healthy tissue.

10. That the cells of the upper epidermis are somewhat larger and more globose than the normal.

11. That the cells of the palisade layer are longer and wider than the normal.

12. That the chloroplastids in the diseased cells are much smaller and fewer in number.

Make DRAWINGS showing (comparatively) the histologic structure of the diseased and normal portions of the oak leaf. Preserve correct proportions between the two drawings.

Can you now explain how the puffing of the lesion is produced?

ETIOLOGY

The pathogene causing the leaf blister of oaks is *Taphrina cœrulescens* (Desmazieres and Montagne) Tulasne, an ascomycetous fungus belonging to the order Protodiscales and the family Exoascaceæ. No fruit body is formed by these primitive ascomycetes. The asci are developed directly from the mycelium in a hymenium on the surface of the leaf. The peach leaf curl pathogene, *Exoascus deformans* (Berkley) Fuckel, is closely related to *T. cœrulescens* (Desm. and Mont.) Tul.

Life History. The life history of *T. cœrulescens* is imperfectly known. It is evidently an obligate parasite. From our knowledge of related fungi, especially that of the peach leaf curl pathogene we may consider it to be about as follows.

The **Primary Cycles** are the only ones certainly known to occur, though the statements of Wilcox (1903 : 173–174) seem to indicate that secondary infections occur in the South at least. Here is an interesting problem for further investigation.

Pathogenesis. The source of inoculum is the diseased leaves on the twigs. Here in late spring or early summer are developed on the convex surface of the blisters a layer of asci containing ascospores. Cut out a small square from one of the larger blisters, chop in a drop of dilute KOH solution on the slide, cover and examine. OBSERVE:—

13. The long hyaline thin-walled sacks,—the asci.

14. The ascospores still within some of them, others empty. Note the open end through which the spores have been discharged.

15. Number, shape, size and color of the ascospores. There are at first 8 spores in an ascus but these bud before liberation forming a large number of smaller secondary spores which are shot out of the ascus and serve as the inoculum. DRAW to show the points observed.

Scattered by the wind some of these secondary spores lodge in the axils of the leaves at the point where the buds for next season are developing.

Here these spores lie dormant attached to the exterior of the developing buds until the following spring. As the buds swell and the young leaves begin to emerge the spores germinate and send their germtubes through and beneath the cuticle. The mycelium branches and spreads in all directions beneath the cuticle. The spreading mycelium irritates the growing leaf tissues causing the histologic changes already observed. From the subcuticular mycelium arise a new crop of asci.

Examine the stained cross-sections of a blister and NOTE:—

16. The mycelium. To what portion of the leaf is it confined?

17. The layer of more or less angular asci standing at right angles to the lower epidermal cells.

18. The wedge-shaped root-like projections pushed down between the lower epidermal cells and which serve as hold-fasts for the asci.

19. The spores within the asci. They are small and either eight in number or numerous (probably in some cases as many as a hundred). Some authors have suggested that the budding of the eight ascospores, which results in these numerous secondary spores without a mycelial stage intervening, constitutes in reality asexual reproduction, and that these spores are conidia.

DRAW a portion of the lower epidermis as seen in prepared cross-sections showing mycelium, hold-fasts, and young and old asci with spores.

Saprogenesis. This fungus apparently has no saprogenic phase, being constantly associated with the living parts of its suscept, throughout its life history.

References

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BLACK KNOT

This is one of the most striking diseases of stone fruits. It is a native disease of our wild species of plums and cherries but is frequently destructive to our cultivated forms, especially certain varieties of plums and sour cherries. The disease is not known to occur outside of North America.

SYMPTOMATOLOGY

Morphologic Symptoms and Signs. The lesions of black knot occur only on twigs, branches, or trunk. The leaves and fruits are never directly affected, nor have galls been reported on the roots of any of the suscepts. Two seasons are required for the full development of the excrescences.

Examine the very young galls provided. Observe:—

1. That they are usually confined to one year old twigs and spurs.
2. Their position with relation to leaf scars or buds.
3. That some have not yet burst through the epidermis while others have, exposing the rough light-colored surface of the young excrescence.

Cut across through one of the galls.

4. In what tissues of the twig does the gall appear to originate?
5. Is the entire circumference of the twig involved in the tumefaction?

Make drawings of young galls in different stages of development; also an outline drawing of the cross-section.

Examine now specimens of the galls collected during June of their first season. Observe:—

6. The larger size of the galls.
7. That young galls on twigs, more than one year old evidently arise at or near one year old spurs or branches in most cases.
8. The fully exposed surface of the excrescence.
9. The rough rim of broken epidermis and cork layer surrounding the gall.
10. The olivaceous felty patches on the exposed excrescence,—the conidial stromata.
11. The brown woody surfaces on which the stromata have not developed.

Study the interior of a knot that has been sawed across and smoothed with a sharp knife. Determine:—

12. What portion of the circumference of the twig is involved in the tumefaction.
13. The thickness of the conidial stroma.

Make drawings of an entire gall and of the cross-section.

Study some of the specimens showing the character of the knots the second season. Observe:—

14. The darker color and more hard and woody character of the galls.
15. The continuous black perithecial stroma covering the exposed surface.

16. The minute pimple-like perithecia arising from, and crowded over the surface of the stroma.

17. Secondary galls arising just above or below some of the primary ones; known by their lighter color and conidial stromata.

DRAW to show the character of mature galls and secondary gall formation.

18. That affected twigs may be bent at right angles. DRAW.

Histologic Symptoms. The effect of the pathogene on the suscept is one of stimulated overgrowth of the invaded tissues. Cut with a knife or scalpel (not a razor) across one of the mature galls (alcoholic material) provided for this purpose. Trim the surface smooth and with hand lens MAKE OUT:—

19. The anatomical distortions that have occurred in the elements of the vascular cylinder and bark, comparing constantly with the structures in the normal portion of the twig.

Make a diagrammatic DRAWING of the entire cross-section showing normal and pathological anatomy. Label corresponding tissues in each.

Study under the microscope the prepared sections through a young gall. OBSERVE:—

20. The character of the tissue on which lies the stroma of the fungus; size, shape, contents of cells and tissue relation.

21. The mycelium in the tissues; inter- or intracellular?

22. The mycelial strands and fans in some places; their pseudoparenchymatous character. What is their relation to the stroma?

23. The depth toward the pith to which the pathological effects are evident.

24. The broadening of the rays in the diseased region; due to hyperplasia or hypertrophy?

25. The isolation of the cambium at the outer ends of the xylem wedges; wanting in the abnormally broad rays.

26. The inhibition of xylem elements and the abnormal development of parenchyma in the xylem wedges. The enlargement of the gall the second year is largely due to a marked hypertrophy of these parenchyma cells.

27. The isolated groups of summer wood vessels in the xylem just inside the cambium.

28. The isolated xylem elements in the diseased bark. These result from the misplaced and broken segments of the cambium opposite the broad rays.

29. The proportional increase in thickness of bark and wood in the diseased region. The outer bark sluffs off before conidial formation.

Compare your findings with those of Stewart (1914).

Make a full page DRAWING, detailing cells in the different regions, to bring out the histological changes emphasized above.

ETIOLOGY

The black knot is caused by the ascomycetous fungus, *Diobotryon morbosum* (Schweinitz) Theissen and Sydow (=*Plowrightia morbosa* (Schweinitz) Saccardo), which belongs to the group known as Pyrenomycetes.

Life History. This pathogene presents in its life history some striking variations from the usual type; it requires two seasons to complete a life cycle and two distinct sets of primary cycles may be initiated each season. True secondary cycles do not occur.

The **Primary Cycles** are initiated in the early spring and early summer by inoculum produced on either one year or two year old galls.

Pathogenesis. The ascospores constitute the inoculum for the first set of primary cycles. With the scalpel remove a few of the mature perithecia and crush in a drop of water or KOH solution on a slide. Cover and OBSERVE:—

30. The ascospores; their form, number in ascus, number of cells and color. DRAW.

If living material is available, study the germinating ascospores. DRAW.

These ascospores mature and are discharged from the perithecia usually during March. They are carried to the opening buds and infect the developing twigs. The development of the pathogene and the reaction of the suscept is slow. Slight swellings on the infected twigs begin to show by autumn. These develop rapidly during the early days of the following spring and burst through the epidermis. On the exposed surface, the scattered conidial stromata develop on which conidiophores bearing conidia are matured in late spring or early summer. These conidia are disseminated and initiate the second set of primary cycles which duplicate in all respects the phenomena of those of the first set.

Study cross-sections through the conidial stromata and OBSERVE:—

31. The structures of the stroma; relation to mycelium in suscept tissues; size, color and septation of the mycelium in the tissues and also in the stroma.

32. The erect conidiophores; septate or non-septate?

33. The conidia; shape, size, color and number of cells; where and how attached to conidiophores?

Make a DRAWING to show these structures.

After the conidia are matured and disseminated, the velvety covering of conidiophores gradually disappears, giving place to the continuous black crust-like stroma of the sexual stage which has been developing, so that by the second autumn the knot shows the black carbonaceous character, distinctive of its maturing condition. The minute pimple-like perithecia (fruit bodies of the sexual stage) are finally developed on the surface.

During the first warm days of the spring (two years after the first primary infections) ascospores are formed within the asci in these perithecia.

Examine with the hand lens, the surface of mature galls provided. OBSERVE:—

34. The minute black bodies crowded together over the surface; shape and size; adherent to each other or separate on the stroma?

35. The apex of the perithecia often depressed; a minute opening at the center of each,—the ostiolum.

Study prepared sections through mature galls showing longitudinal sections of perithecia. MAKE OUT:—

36. The relation of perithecia to each other and to the stroma; character of the stroma as compared with that of the conidial stage.

37. The structure of the perithecial wall; ostiolum; relation of asci and paraphyses to the perithecial cavity.

Crush in water one of the mature perithecia. OBSERVE:—

38. The structure of the asci, ascospores and paraphyses.

Make an enlarged DRAWING of a longitudinal section of the perithecium and supporting stroma to show these structures in detail.

The ascospores mature in early spring and are shot, during rain periods, from the asci through the ostiolum. Caught by the breeze, they are carried to the trees and infection occurs in the swelling buds or developing shoots. Thus, in two full years is the primary life cycle, starting with ascospores, completed in the production of another crop of ascospores.

Saprogenesis. As the pathogene is continuously associated with its living suscept, it may be regarded as having no saprogenic phase in any of its life cycles.

Secondary Cycles. True secondary cycles (resulting from infection by spores produced during the season in which primary infection takes place) do not occur. The development of secondary galls by mycelium spreading from the margin of old galls into the adjacent tissue is however regarded by some workers as secondary infection (Stewart 1914 : 114).

References

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CORN SMUT

This is the most common and most noticeable disease of corn. It is sometimes known as boil smut to distinguish it from the head smut. It affects all varieties of field corn, pop corn and sweet corn. Some varieties of sweet corn suffer severely from this disease.

SYMPTOMATOLOGY

The **Signs** and **Morphologic Symptoms** of this disease will be considered together. All parts of the plant above ground are subject to infection. Unlike most of the cereal smuts, invasion is local. The lesions are characterized by marked hyperplastic enlargements.

On the stalks. Study the specimen provided and OBSERVE:—

1. At what points on the stem the galls are usually located.

2. The large size of the boils and the comparatively small area from which they arise. Is there any evidence of injury to the adjacent tissues of the stem?

3. The texture of the boils; the tough fleshy covering of young galls. The boils are more firm in fresh specimens, especially when yet immature.

4. The color; compare old and young boils.

5. The dark dusty spore mass filling the mature boils.

A relatively large portion of the gall is composed of pathogene structures (mycelium or spores).

Make a SKETCH to show various forms of stem boils.

On the leaves. Lesions are comparatively rare on the leaves. In the specimens provided, OBSERVE:—

6. The small boils arranged in rows parallel with the veins. Do they seem to arise from or between the veins; which side of the leaf?

7. Any effect on the tissues adjacent to the boils; opposite the boils. The sooty spore mass within the galls.

SKETCH leaf lesions.

On the ear. Affected ears are very common, especially those with exposed tips. In the specimens provided, OBSERVE:—

8. The form, size and location of the boils.

9. That they are each an enlarged kernel. Find kernels in different stages of tumefaction.

SKETCH a diseased ear to show healthy kernels and diseased ones, in various stages of tumefaction.

On the tassel. The boils of this smut are very common on tassels but may be confused with those of the head smut. (See Hitchcock and Norton 1896 : 199 and pl. 6, 8–10.) In the specimens provided, OBSERVE:—

10. That the boils on the tassels are but malformed parts of the flowers.

11. That only flowers here and there in the tassel are affected.

12. That the character and degree of malformation varies in different flowers.

Carefully dissect apart a healthy and a diseased flower. Make a series of comparative DRAWINGS to show the effect of the disease on the different organs of the male flower.

For a study of **Histologic Symptoms** examine prepared sections through young kernel galls in comparison with sections through young healthy kernels. DETERMINE:—

13. Modifications in the epidermis and seed coat cells due to the disease.
14. Histologic modification of the other tissues of the kernel.

Compare your findings with those of Knowles (1889).

Make detailed DRAWINGS to show histologic modifications.

ETIOLOGY

The pathogene causing corn smut is *Ustilago zeæ* (Beckmann) Unger, one of the Ustilaginaceæ. It was first described as one of the puff balls by Beckmann in 1768 and has been, since that time, a frequent subject for investigation by mycologists and phytopathologists.

Life History. It differs markedly in its life habits from most of the common cereal smut pathogenes. There are normally, in temperate climates at least, only the primary cycles.

The **Primary Cycles** are initiated at any favorable time during the period of active growth of the corn. Overwintered chlamydospores, usually in the manure on the field, are the sources of inoculum.

Pathogenesis. Germinate some of the chlamydospores from an old overwintered smut boil in manure extract on slides. Examine under the microscope and OBSERVE:—

15. The large globose spiny dark brown chlamydospores.
16. From some of these, the protruding hyaline promycelium (basidium).
17. The crack in the epispore through which the promycelium is protruded.
18. The septa in the promycelium, from below each of which arise one to several slender, pointed sporidia. They are often produced in great numbers; sometimes in chains.

DRAW a germinating chlamydospore showing abundant sporidial production.

These sporidia are carried by the wind to the growing corn. Some of them lodge on the growing tissues of the suscept near the joints within the leaf sheath, the emerging silks, or the blossoms on the tassels. Here they germinate sending out germtubes that penetrate these embryonic tissues, and give rise to the mycelium. This mycelium does not spread through the tissues to any great distance from the point of invasion; every boil probably arises from a separate infection. The hyphæ grow in great masses into the tumefying tissues of the boil and form the greater bulk of the excrescence. Chlamydospores are soon developed in chains within short branches of this mycelium. The walls of the mycelium gelatinize and disintegrate more or less.

To study the mycelium in the tissue about the base of the boil, make thin longi-sections of the stalk, just beneath the boil. Stain with methyl blue or eosin. Examine and in the pith tissues OBSERVE:—

19. The slender hyphæ in the intercellular spaces or penetrating the cells; often in strands of several hyphæ or gnarled in the intercellular spaces.

20. Short knotted or irregularly swollen short branches sent into the cells,—the haustoria.

DRAW to show the mycelium in the tissue.

To study the distribution of the mycelium in the boil and the formation of the chlamydospores, examine prepared sections through a partly matured boil. Make DRAWINGS to show formation of spores.

Saprogenesis. Mount some of the black spore mass in KOH solution. Study with the high power and OBSERVE:—

21. The shape, size and color of the mature chlamydospores; the short spines which thickly beset the spore walls.

22. Any variation in the spores as to size and shape. Look for bits of mycelium.

Make an enlarged DRAWING of several of the spores.

These chlamydospores go with the fodder into the manure and finally out on the land. They winter over in the manure and germinate during the following season. Their dependence upon saprophytic nourishment in germination is evidenced by the fact that they develop sporidia much better in a nutrient solution, e. g., manure extract than they do in water.

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HOLLYHOCK RUST

THIS is a rust disease peculiar to a number of malvaceous plants. It is indigenous to Chili where it was discovered in 1852. It is especially destructive to the hollyhock and is now known as a common disease of this perennial in nearly every temperate country of the globe.

SYMPTOMATOLOGY

The **Signs** and **Morphologic Symptoms** of this disease may best be studied together. The lesions occur on all the above-ground parts of the suscept, but are usually most numerous on the leaves. In severe cases, the calyx and seed capsules are also affected.

On the leaves. Examine the specimens provided and OBSERVE—

1. The cushion-like pustules scattered over the leaf surface,—the sori of the pathogene; most abundant on which side of the leaf?

2. The variations in size and shape of the sori.

3. Color of the lesions. This varies with age. The youngest lesions usually show a distinct bright yellow color, especially on the upper surface or where they occur on the veins and petioles. A little later the fully developed sorus (telium) takes on a rusty brown color and is surrounded by a yellowish zone. When the spores of the telium begin to germinate, the color of the sorus becomes a grayish brown. As the pustule dies it turns black.

4. The thickened character of the leaf tissue in well developed lesions; there is slight hypertrophy.

5. In old lesions, the dead and shriveled sorus surrounded by a narrow zone of necrotic leaf tissue. This dead tissue sometimes falls out, leaving small round holes in the leaf blade.

DRAW a diseased leaf to show the characters of the lesions at various stages.

Where the lesions are very numerous, the entire leaf blade withers, turns brown and falls prematurely from the stem. The general effect of a severe infection is best observed in the field.

On the stems and petioles. Examine the specimens provided. OBSERVE:

6. The shape and size of the pustules; usually larger than those on the leaves. Why?

7. The elongated dead zone about the older lesions. How deep does the necrosis extend? The lesions are very limited. They frequently die out and disappear except for a large oval scar left by the healed wound.

DRAW several stem lesions of different ages.

On the fruit. Study the lesions on the flowers provided (best studied on the fruits of *Malva rotundifolia* L.). OBSERVE:—

8. The lesions on the calyx; compare with those on leaves and stems.

9. The sori on the seed carpels; color, size and location. DRAW. The pathogene is often distributed on seed infested in this manner.

The **Histologic Symptoms** may be studied in the sections (free-hand or prepared) through a young lesion. Study under the microscope and DETERMINE:—

10. The modifications in cell contents which account for the yellow color of the lesions.

11. In what tissues the hypertrophy occurs. Is there also some hyperplasia?

Make a detailed DRAWING of the section to show histologic modifications.

ETIOLOGY

The hollyhock rust is caused by a uredinaceous fungus, *Puccinia malvacearum* Montagne.

Life History. It is a so-called lepto-form or Leptopuccinia, i. e., it has neither æcia nor uredinia, producing only telia. The teliospores usually germinate *in situ*, as soon as mature, to form promycelia with sporidia.

The **Primary Cycles** occur on the early leaves developed from the perennial root in the spring. The sources of inoculum are the old dead leaves, diseased the previous season, or overwintered living leaves in which the pathogene has hibernated.

Pathogenesis. The inoculum consists of the sporidia. These are formed on the promycelium which develops from the teliospore. Teliospores which are produced late in the autumn often do not germinate at once but remain dormant on the old dead leaves. More commonly, however, late infections on immature green leaves of the hollyhock or on those of *Malve rotundifolia* do not develop telia until early spring. The teliospores in these early telia germinate and sporidia for the primary infections are formed.

Study germinating teliospores; or illustrations in Phytopath. **1**, pl. 13. OBSERVE:—

12. The long slender basidium (promycelium), most commonly developed first from the apical cell.

13. The densely granular protoplasm of the apical portion of the basidium; its final division into four cells, and the formation of a sporidium from each.

14. That sometimes the four apical cells separate before sporidia are developed.

Make a series of DRAWINGS to show sporidial formation.

The sporidia are carried by the wind or splashing rain to the leaves nearby. Here they quickly germinate sending forth a short germtube which penetrates the epidermis of the suscept.

DRAW several germinating sporidia. These sporidia are uninucleate and develop within the tissues a septate, uninucleate mycelium. Study thin sections (free-hand or prepared) through a lesion. OBSERVE:—

15. The slender, often matted, septate mycelium in the intercellular spaces forcing the leaf cells apart. Are haustoria formed?

16. The matted mycelial stroma from which arise the slender teliospore stalks, forming the telial sorus.

17. The teliospores; form, size, solor, septation and thickness of walls.
18. Young spores in various stages of development.

Make a detailed DRAWING of a telial sorus in section with adjacent leaf tissue and intercellular mycelium.

These teliospores germinate *in situ*, giving rise to the sporidia which, scattered by wind or rain, initiate secondary cycles.

Saprogenesis does not occur in the primary cycles.

Secondary Cycles are initiated repeatedly throughout the season and conform in their development to that of the primary cycles except that in the case of late secondary cycles, teliospores which have developed, may fail to germinate at once and overwinter on dead leaves and stems. Very late secondary cycles on overwintered living leaves may fail to develop telia until the following spring. Saprogenesis in secondary cycles is limited to the germination of overwintered teliospores on dead débris of the suscept.

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RUST OF CEDAR AND APPLE

This is a very common disease of the apple in many sections of the country where the red cedar grows. It is especially prevalent in the Mississippi Valley, eastern New York, New England, Virginia, West Virginia and other southern states. The effect of the pathogene on both suscepts is very marked. Not all varieties of apples are susceptible. The rusts appearing on pear, quince and hawthorne are usually caused by pathogenes distinct from that causing the apple rust; all, however, are closely related.

SYMPTOMATOLOGY

The **Morphologic Symptoms** of this disease differ strikingly in the alternate suscepts.

On the *apple tree* the lesions are usually confined to the leaves and fruits. Twigs and fruit spurs are occasionally affected (Reed and Crabill 1915, fig. 12 and Weimer 1917, 515).

Leaf lesions are most common. They are often sufficiently numerous to give the trees a distinct yellow appearance even at some distance from the orchard. In the specimens provided OBSERVE:—

1. The color of the lesion on the upper side of the leaf; at first pale yellow finally becoming orange, often with a red margin.

2. The slight hyperplastic thickening of the leaf on the under side of the lesion, best detected in fresh material.

3. The size of the lesions; definiteness of delimitation and color on the two sides of the leaf.

The **Signs** of the disease on the leaf are diagnostic. Observe:—

4. The presence of black pimple-like structures on the upper surface of the lesion,—the pycnia (spermagonia) of the pathogene, probably functionless male structures. These are at first amber yellow in color.

5. The groups of brownish, fimbriate cup shaped structures on the lower surface,—the æcia (æcidia) or cluster cups.

Sketch to show the lesions as they appear on both sides of the leaf.

Defoliation frequently results from a severe leaf infection. (Giddings and Berg 1915, pl. 7).

Fruit lesions begin to appear usually when the apples are about one-fifth grown but do not fully develop until near the end of the season. Examine the specimens provided and OBSERVE:—

6. The color and location of the lesions.

7. The dwarfing effect on the fruit. Recall the effect on the leaf.

The **Signs** of the disease on the fruits are identical with those on the leaves except that both pycnia and æcia are formed on the same surface, the latter following the former in a zone around the pycnial area.

Make enlarged DRAWINGS to show the surface of a mature fruit lesion.

On the *cedar* the lesions produced are tumefactions,—true galls.

Leaf galls. Examine full grown galls collected in the winter or early spring. OBSERVE:—

8. Their form, size, color and attachment to the twig.

9. Note their great variation in size. (See Giddings 1918, pl. 5 and illustration specimens.)

SKETCH a branch showing galls of various sizes. Examine the surface of a large gall and NOTE:—

10. The small circular pits scattered over the surface (Jones and Bartholomew 1915, fig. 4). It is at these places that the spore horns (telia) of the pathogene break forth the following spring.

Make an enlarged DRAWING of a gall.

Each gall results from the monstrous development of a single leaf of the cedar. (Weimer 1917 : 513.) Remove one of the healthy leaves and NOTE:—

11. Its size, form and relation to the twig.

SKETCH a normal leaf and a diseased or galled leaf. (See Heald 1909, pl. 1, fig. 1 and pl. 12.)

The most striking **Sign** of this disease on the red cedar is exhibited during warm spring rains in April and May. From the telial stromata scattered over the gall, long yellow gelatinous teliospore masses protrude, giving the effect from a distance, of large yellow fruits whence the popular names, cedar-apples and cedar-flowers. (See Weimer 1917, fig. 137 and 138.) When these horns first break forth they are short pointed and of a chocolate brown color. Study a gall in which the horns have just protruded. SKETCH a horn and adjacent gall surface enlarged about 4 times.

With the warm spring rains of April and May these horns rapidly elongate and gelatinize. Examine such a gall and OBSERVE:—

12. The length, shape, and consistency of the gelatinized horns. They are of a bright yellow color in fresh specimens.

SKETCH a gelatinized telial horn with adjacent gall surface enlarged about 4 times. This is the mature telium.

The **Histologic Symptoms** are primarily hyperplastic in the lesions both on the apple and on the cedar.

In the apple leaf. Examine free-hand or prepared sections through a lesion and OBSERVE:—

13. The hypertrophy and hyperplasia of the mesophyl cells and the obliteration of the intercellular spaces.

14. That the palisade cells are little affected as to size and numbers. Can you detect any other changes? (Compare Reed and Crabill 1915, fig. 13.)

DRAW a portion of the section to show histologic changes.

In the cedar leaf. Since the gall is but an enlarged leaf, study thin sections of a healthy cedar leaf and MAKE OUT:—

15. Its structure. (See Reed and Crabill 1915 : 18.) DRAW.

In order to understand the gross modifications of the leaf in the production of the gall, study thin longi-sections of a young gall or see Reed and Crabill (1915, fig. 4). DRAW a diagram of a longi-section through a young gall and adjacent healthy leaves.

Make thin sections through a large gall and MAKE OUT:—

16. The outer rind or cortex; its color. How many cells in thickness?

17. The parenchyma, large oval thin walled cells making up the bulk of the gall; cell nuclei. How many in each cell; starch grains.

18. The much branched and distorted vascular system embedded in the parenchyma (Reed and Crabill 1915 : 20–21).

DRAW a portion of the section to show the histologic structure of the gall.

ETIOLOGY

This disease is caused by a basidiomycete, one of the Uredinales or rust fungi, *Gymnosporangium juniperi-virginianæ* Schweinitz. The pycnial stage (0) and the æcial stage (I) occur on the leaves and fruits of the cultivated apple and wild crab. The telial stage (III) is found on the red cedar. A uredinial stage (II) is wanting.

Life History. The apple rust fungus continues in close association with the living tissues of its suscepts throughout its entire life history. There is no saprogenesis in any of its cycles.

Primary Cycles occur on the apple leaves, fruits and twigs.

Pathogenesis. The inoculum for the primary cycles consists of sporidia which are produced on the galls on the red cedar. They are developed on a promycelium which in turn arises from a teliospore. Moisture of six or more hours' duration is necessary for sporidial formation. As soon as the humidity decreases sufficiently to cause appreciable evaporation, the sporidia are forcibly ejected. This may occur with rains at any time from April to June. Sporidia are carried to the infection courts by the wind. Germination follows within a short time in the presence of moisture. Two types of germination occur; one in which germtubes develop directly from the sporidia; and the other in which secondary sporidia are formed on a short germtube. The latter type is more common. (See Phytopath. **3** : 282, fig. 1 and Reed and Crabill 1915, fig. 8–9.) DRAW.

The germtube arising from the sporidium penetrates the cuticle on the upper surface of the leaf. The mycelium developed from the sporidial germtube spreads through a limited area of the leaf, with the result that the tissue changes already observed are effected and the characteristic fruit bodies are developed. Examine the apple leaves again, using the hand lens. OBSERVE:—

19. The pycnia on the upper surface of the leaf; their form and distribution.

20. The æcia on the lower surface of the leaf; their form, distribution and size. The long exerted peridium (æcial wall),—a prominent character of the æcia of this fungus. Rust fungi showing æcia with such peridia belong to the form genus, Rœstelia. Accordingly the apple rust fungus was formerly called *Rœstelia pyrata* (Schw.) Thaxter. (See Heald 1909, pl. 9.)

Make enlarged DRAWINGS to show the pycnia and æcia as they appear under the hand lens.

Mount in KOH solution, some of the peridial cells and æciospores. Study and DRAW to distinguish them.

Examine prepared slides showing sections through lesions on an apple leaf. OBSERVE:—

21. The pycnia; their form and contents.

22. The æcia; their position with reference to the pycnia; the relative size of the two structures; their form, structure and contents; the æciospores borne in chains on the ends of short stalks arising from the base of the cup.

Make a DRAWING to show the points observed in 21 and 22. (See Reed and Crabill 1915, fig. 13.)

The pycnial stage always precedes the æcial stage. Æciospores never reinfect the apple.

Secondary Cycles occur on the red cedar only.

Pathogenesis. The inoculum for the secondary cycles consists of the æciospores which are discharged from July first to the end of the growing season. They are blown to the cedar and lodge in the axils of the leaves. The æciospores apparently do not germinate at once. The question of whether infection occurs in the fall or spring is yet unsettled. (See Reed and Crabill 1915 : 49–51.) The way in which the germtube gets into the cedar leaf is not known. The manner of germination is not essentially different from that of other fungous spores. A simple germtube is formed. This on entrance into the leaf tissues develops into a mycelium which stimulates the growing cells and a gall results. This becomes evident the following June.

Study the young green galls provided. These increase in size during the summer and become brown and full sized by autumn. The fungus passes the second winter of its life as mycelium in the full-sized gall. From one of the galls provided, remove a small bit of tissue. Cook for a few minutes in weak KOH solution. Mount in a drop of water on a slide and crush. Stain a few minutes with eosin, wash out with water and OBSERVE:—

23. The hyphæ throughout the mount between the parenchyma cells. Are they septate or continuous?

Examine prepared sections through mature galls. NOTE:—

24. The mycelium of the fungus in its relation to the parenchyma cells. Are haustoria present? (See Reed and Crabill 1915, fig. 5.)

In the autumn the mycelium forms stromata at certain places beneath the cortex, stromata from which the telial horns will be developed the following spring.

Remove one of the gelatinized horns from a gall, split it lengthwise, mount in a drop of water on a slide, cover and crush it out by gently pressing on the cover glass. OBSERVE:—

25. That it is made up of long slender hyaline stalks each bearing at its tip a two-celled teliospore (teleutospore). (See Reed and Crabill 1915, fig. 7.)

Make an enlarged DRAWING of several of the teliospores.

The teliospores germinate during the spring showers as we have seen. As the horns dry following the shower the basidiospores are shot off from the sterigmata and carried by the wind to the apple leaves thus completing the life history.

Repeated wetting of the telial horns finally results in the germination of all the teliospores. The galls then shrivel and fall. Occasionlaly a secondary growth of the gall takes place so that it persists until another season.

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BLISTER RUST

THE blister rust of five needled pines and its associated stages on currants and gooseberries, has now become an important disease in the United States. Previous to the importation of diseased white pine nursery stock, this disease was confined to Europe. Recently it has assumed an epiphytotic character in several localities in northeastern United States.

SYMPTOMATOLOGY

The **Signs** and **Morphologic Symptoms** of this disease will be studied in the seasonal sequence in which they appear on the alternate suscepts.

Blister rust stage. This phase of the disease occurs only on pines. In eastern United States it is best known on white pine.

In the illustrations and material provided, OBSERVE:—

1. That the first evidence of the disease is an indefinite discolored canker-like area on the trunk or smaller branches (Colley 1918, pl. 48).

2. That in some cases a slight to marked swelling of the bark occurs. DRAW or COPY.

3. That often a diseased tree may be detected by its stunted growth and bushy appearance (see Farmers' Bul. 742, fig. 1).

4. That the first signs of the disease are the minute light yellow, bladder-like swellings which exude drops of liquid. These small pustules are pycnia (spermagonia). They first appear several months after infection and during the autumn just previous to the production of æcia.

5. The large hemispherical or pustular bladdery æcia pushed out from cracks in the bark.

6. The æcia, at first yellow and enclosed by a white papery peridium; later irregularly ruptured, allowing the yellow powdery contents,—the æciospores, to escape. The æcia are produced in early spring two or more years after infection.

7. The pock-like depressions in the bark where æcia have been borne. These characteristic markings on the cankered area constitute a reliable means of identification of diseased trees at any time of the year after the first crop of æcia has been produced.

DRAW cankers to show typical signs and symptoms.

Orange rust stage. The æciospores from the lesions on the pine infect the young leaves of many species of Ribes producing symptoms very different from those on the pine. Examine the diseased leaves of the different species of Ribes provided. OBSERVE:—

8. That in the case of certain species, definite pale green lesions are produced; in others the fruit bodies of the pathogene alone constitute the first evidence of the disease. DRAW.

9. The very small hemispherical pustules,—the uredinia, in groups on the under sides of the leaves. They are at first covered by the epidermis and are different in color from the remainder of the leaf. DRAW a leaf showing the lesions as seen with the hand lens.

10. That later the uredinia become more or less orange yellow in color; the covering (composed of leaf epidermis and peridium) is ruptured allowing the rusty yellow contents,—the uredospores, to escape.

Felt rust stage. In the material of gooseberry and currant leaves collected later in the summer NOTE:—

11. The conspicuous hair-like growths,—the telia, often arising from the uredinia and more or less scattered over the entire under surface of the leaves. DRAW a portion of the diseased leaf as seen under the hand lens.

In some cases the felt rust stage accompanies the first uredinia early in the summer but it is typically an autumnal stage, occurring in August and September.

[ETIOLOGY

The blister rust is caused by the basidiomycetous rust fungus, *Cronartium ribicola* Fischer von Waldheim. This name is based on the telial stage. The æcial stages of rusts, forming blister-like æcia on the bark or needles of conifers, have all been placed in the form genus Peridermium. The blister rust of five needled pines is commonly known in its æcial stage as *Peridermium strobi* Klebahn.

Life History.—This pathogene is a heterœcious parasite requiring two very different hosts for the development of its complete life history. All the spore stages produced by rusts are formed by this fungus: æcia (I) on bark of pines; uredinia (II), telia (III) and promycelia (IV) on the leaves of currants and gooseberries. The pycnia (0) are produced on the pine previous to the appearance of the æcia.

Primary Cycles are initiated by the æciospores produced on the pine in early spring. When the peridium is ruptured, the æciospores dust out and are blown about by the wind.

Pathogenesis. The æciospores must reach young leaves of currants and gooseberries in order to function in continuing the development of the fungus. The æciospores cannot infect the pine.

Mount some of the yellow powder from the æcia in KOH solution, cover and examine with the microscope. EXAMINE:—

12. The size, shape and color of the æciospores; thickness and markings of the spore wall. DRAW.

The æciospores in the presence of moisture, germinate on the under sides of gooseberry or currant leaves and the germtubes enter through the stomata into the sub-stomatal cavities (Colley 1918 : 634). After the establishment of a food relation with the leaf cells surrounding the sub-stomatal cavity, a mycelium is produced which spreads in a limited area. Within about two weeks after infection the uredinia are formed. In the material studied under symptoms of this stage, NOTE:—

13. The general character of the uredinia in different stages of development.

14. The size, shape and color of the uredospores under the microscope; contents.

15. The thickness and markings of the spore wall.

DRAW a typical uredospore showing above points.

In the prepared slides of stained sections through uredinia, NOTE:—

16. The epidermis of the leaf which was raised up and finally ruptured by the developing uredinium.

17. The peridium enclosing the contents of the uredinium (Colley 1918, pl. 55 C).

18. The sporophores each bearing a single uredospore. How many nuclei in each spore?

Make a DRAWING of the uredinium in section as seen under the microscope.

The first production of uredospores furnishes inoculum for one type of secondary cycles, which are repeated several times during the summer on currants and gooseberries. Both uredinia and telia are produced in these secondary cycles which end with the falling of the leaves in the autumn.

In some cases the telial stage is produced from the same mycelium which produced the first crop of uredospores, and thus the primary cycle may be carried forward to the production of sporidia by the germinating teliospores.

In the prepared slides showing uredinia in section, NOTE:—

19. The stroma developed just beneath the uredinium and the radiating mycelium extending into the leaf tissue. Find the haustoria. DRAW.

In prepared slides showing the telial stage, OBSERVE:—

20. That the telial horns arise directly from the same or a similar stroma to that which bore the uredospores.

21. The telial horn, composed entirely of teliospores cemented together in a compact mass (Colley 1918, pl. 52 C). DRAW.

The teliospores germinate *in situ*. Mount telial horns bearing germinating spores (if available) or study photographs and drawings (Colley 1918, pl. 56C and 57; Clinton and McCormick, 1919 pl. 39 and 44). OBSERVE:—

22. The short often curved promycelium produced from each teliospore; septation, sterigmata, sporidia.

DRAW to show teliospore germination and sporidial formation.

Thus ends the primary cycle. There is no saprogenesis.

Secondary Cycles. As already pointed out uredospores initiate secondary cycles on the leaves of Ribes and these may be repeated several times during the season finally resulting in the formation of telia during summer and autumn. The telia whether produced from primary or secondary lesions on Ribes leaves furnish the inoculum (sporidia) for another set of secondary cycles, namely those on the white pine.

These sporidia (probably forcibly discharged from the tips of the sterigmata) are carried by the wind to the leaves of pine trees nearby. This appears to occur most abundantly in late summer and autumn. Here the sporidia germinate (Clinton and McCormick 1919, pl. 44; Colley 1918, pl. 57) and send their germtubes through the stomata into the leaf. Immediately on passing through the stomatal opening the germtube swells to form a substomatal vesicle (Clinton and McCormick 1919, pl. 43, fig. 1–6, g) from which a single mycelial thread is produced. Study the illustrations referred to and OBSERVE:—

23. The branching and development of the mycelium in the substomatal cavity.

24. Formation and character of haustoria.

25. The formation and structure of the so-called sclerotia formed in the leaf (Clinton and McCormick 1919, pl. 43, fig. 7 and pl. 41, fig. 3).

Make a DRAWING of a section through the pine leaf to show invasion and infection of the pine leaf. Read Clinton and McCormick (1919 : 447–455).

The fungus slowly spreads along the vascular strand in the leaf down into the bark of the stem eventually causing the typical bark canker of the blister rust stage during the following and succeeding years, on which are produced first the pycnia in the autumn and then æcia the following spring. In these æcia are produced the æciospores which serve as inoculum for another set of primary cycles.

From the material of affected pines, take a bit of the peridium; stain with eosin and mount in water. OBSERVE:—

26. The character of the cells of the peridium. DRAW.

Study the sections through white pine twigs bearing æcia. NOTE:—

27. The position of the æcia in relation to the tissues of the twigs.

28. The stroma at the base of the æcium and the radiating mycelium in the bark tissue; the haustoria in the cells (Colley 1918, pl. 53.)

29. The sporophores each bearing a chain of æciospores. DRAW in detail an æcium with the surrounding bark tissues.

The mycelium in the canker may continue to live and enlarge the lesion for years, especially in the large limbs or trunk producing a crop of æciospores each spring.

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THE NEMATODE DISEASE OF WHEAT

THIS disease, long known in Europe, was first discovered in the United States in 1917–18 (Byars 1920). It appears to be most common and destructive in the Atlantic Seaboard states, from Virginia south though it occurs to a limited extent north of this. It is most destructive in wheat and rye, but may affect oats, spelt and emmer to a limited extent (Phytopath **9** : 283–284).

SYMPTOMATOLOGY

The **Morphologic Symptoms** of the disease are so characteristic as to make its identification even in the growing plants certain and easy. The aërial organs of the suscept only are affected.

In the material provided OBSERVE:—

1. The dwarfing of affected plants. This is not always marked.

2. The crooking of the culms at the nodes.

3. The wrinkling and twisting of the leaves. The affected leaves are sometimes tightly twisted into a spiral roll. (Fromme 1919, fig. 1.)

4. The small raised rounded galls on the upper surface of the leaves. Best seen in growing young leaves.

5. The yellowing, wilting and dying of the leaves in the advanced stages of the disease.

See Byars (1920 : 6–8) for detailed description of leaf and stem symptoms. Make SKETCHES to show these symptoms.

Examine diseased wheat heads and OBSERVE:—

6. That they are short and thick as compared with the healthy heads. They mature later than healthy heads and so remain green for some time after normal heads begin to ripen.

7. That the diseased ovaries at flowering time are larger and of a darker green than those in the healthy spikelets. The ovaries become galls.

8. That in the mature diseased heads the galls are shorter and thicker than the healthy kernels. Compare as to color.

9. The superficial similarity of the nematode galls to the smut balls in wheat affected with the stinking smut. How may they be readily distinguished.

Read Byars (1920 : 8) and see pl. 3–4. Make DRAWINGS to show the characters of the nematode kernel galls; also smut balls of stinking smut.

The distinguishing **Sign** of the disease is to be observed only under the microscope. Crush one of the kernel galls (after soaking) in water on a slide and examine under the low power. OBSERVE:—

10. The numberless long thread-like worms which constitute the bulk of its contents. These are the nematode worms which cause the disease (Byars 1920, pl. 5–6). Their presence in the lesions in leaves or heads is the final test of the identity of the disease.

The **Histologic Symptoms** are both hypoplastic and hyperplastic in character. To study the leaf galls make thin cross-sections and examine under the microscope. DETERMINE:—

11. What tissues of the leaf are affected.
12. Cell modifications, their nature and extent.

The modification of the floral organs appears to be confined to the ovary and stamens, chiefly the former. Read Byars (1920 : 9–10) and see to what extent you can verify his findings as to the origin and structure of the flower galls.

ETIOLOGY

The cause of the eel worm disease of wheat is a nematode, *Tylenchus tritici* (Steinbuch) Bastian. It was discovered and described by an Englishman, Needham in 1743, who however, did not give it a name. Steinbuch who first named it called it *Vibrio tritici*. It was finally placed in the genus Tylenchus by Bastian in 1864 (See Byars 1920 : 2–3).

Life History. The causal relation of *Tylenchus tritici* to the disease has been fully established by several investigators. The following studies will make clear the main facts in its life history.

Primary cycles. The pathogene overwinters in the débris of the diseased plants in the soil and in the kernel galls mixed with the threshed and stored grain.

Pathogenesis. The source of the inoculum for the primary cycles is usually the galls mixed with the sown grain. Crush a bit of one of the galls in a drop of water on a slide and study under the microscope. OBSERVE:—

13. The thread-like larvæ floating in the water in great numbers. These larvæ are in the so-called second stage having already moulted once or twice.

14. That they soon become active. Note their eel-like movements.

15. Under the high power, the form and size of the larvæ. Determine relative length of the worm in comparison with its thickness.

16. The relatively simple structure, an outer tube or body wall and an inner tube, the digestive tract (Compare Byars 1920, fig. 3).

17. The mouth at the blunt anterior end and the anus or vent at one side a short distance back from the pointed posterior.

18. The buccal spear at the anterior end of the digestive tract. Controlled by strong muscles this may be exerted, enabling the larva to pierce the egg membrane in escaping from the egg. Look for the empty egg shells. It is by means of this spear also that it can gain entrance into the tissues and extract the sap from the cells. The spear is traversed by a very slender tube.

19. The muscular esophageal bulb a short distance below the base of the spear. By the expansion and contraction of this bulb the fluids are pumped out of the cell into the esophagus through the spear.

20. The posterior esophageal bulb at the base of the esophagus.

21. The large tubular intestine comprising the remaining three-fourths of the alimentary tract. Its oily globular contents, probably reserve food.

22. The body cavity, practically transparent.

23. The half-moon shaped hyaline body about midway the length of the body, the primordial reproductive body.

Make a detailed SKETCH of one of these larvæ to show the structures studied above.

The larvæ in this stage may live for several years in a desiccated condition, becoming active again when placed in water. These larvæ constitute the inoculum. They undergo no further development until they gain entrance into the living suscept.

By their eel-like locomotion they travel through the soil after liberation from the galls and reaching a wheat or other suscept seedling they congregate within the leaf sheath near the growing point. They do not appear to enter the roots. Some of them penetrate the leaf tissues but most of them lead an ectoparasitic life inside the leaf sheaths near the growing point until the head begins to form. They now penetrate the outer cells of the flower fundaments and stimulate cell growth which results in gall production.

Section one of the young flower galls and OBSERVE:—

24. The central cavity containing the worms.

25. The long slender males and the stout females. (Compares Byar 1920, fig. 5 and 6.) How many in each gall?

26. The capsule-shaped eggs; their granular contents. Note that they are one-celled. These eggs begin to segment shortly after they are laid, the young larvæ quickly develop and escape within a few days into the gall cavity where after one or two moults they go into a dormant condition with the maturing of the galls.

27. Young larvæ in the gall cavity (see Byars 1920, fig. 3). Read Byars (1920 : 16–19) and compare your findings with his description of male and female worms.

Make DRAWINGS to show the eggs, young larvæ and mature worms with such stages in development as you were able to discover in the material available.

Saprogenesis. Although the larvæ are capable of remaining alive in the soil for some months there is no evidence that they can feed or develop therein.

To what extent secondary cycles occur if at all is not clear. They probably do not normally occur.

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EDEMA

EDEMA is a physiogenic disease due to a plethora of water in the plant. Although seldom of importance under field conditions it sometimes becomes destructive in the greenhouse. The term intumescence is commonly applied to the lesions of edema, as well as to similar tumefacient lesions sometimes produced by toxic substances applied to the surface of leaves. Many kinds of plants are subject to this disease (Küster 1916).

SYMPTOMATOLOGY

The **Morphologic Symptoms** of edema may be observed in growing tomato, potato or cabbage plants held for some days under a bell jar. Examine the material provided and OBSERVE:—

1. The tumid character of the lesions.
2. Their distribution over the affected organs.
3. The color of the intumescences.
4. Variations in size and shape.
5. The rough scabby character of the old lesions.

Make DRAWINGS to show these symptoms.

The **Histologic Symptoms** may be readily investigated in thin sections (free-hand or prepared) through young leaf and stem lesions. Examine such sections under the microscope and OBSERVE:—

6. The marked hypertrophy of the cells forming the intumescence. What tissues are affected?
7. The almost complete absence of chloroplasts in the enlarged cells.
8. Effect on the cytoplasm and nucleus of the enlarged cells.
9. The presence of crystalloides or other bodies in the hypertrophied cells.
10. The thin walls of the hypertrophied cells.
11. Effect on the epidermal cells and stomata.
12. Whether there is any evidence of hyperplasia.

Make DRAWINGS to show the histologic modifications exhibited in the sections.

ETIOLOGY

No pathogene is responsible for the disease. It is caused by factors which produce abnormal turgidity in the epidermis, spongy parenchyma of the leaves or the cortical tissues of the stem, such as excessive absorption of water by the roots and reduced transpiration from the tops.

Read the article referred to below.

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LABORATORY TECHNIQUE

DIRECTIONS FOR GERMINATING SPORES

A STUDY of germinating spores of the pathogene will be desirable in the case of several diseases as for example, Brown rot of stone fruits, Late blight of potatoes, Botrytis blight of peonies, Bean anthracnose, Cercospora leaf spot of beets, Apple scab and others.

Each student will set up his own spore germination apparatus. It will be well to look ahead and put the spores to germinate a day or two before they will be required for study.

Proceed as follows:

1. Prepare moist chambers (one or more) using petri dishes with two narrow strips of glass in each.
2. Turn the dish upside down making the cover the bottom. Place the glass strips, (a), parallel at opposite sides of the dish, as shown in the accompanying illustration.
3. Flame a clean slide, (b), and place it on the glass strips.
4. Place a drop of clean tap water, (c), in the center of the slide.
5. With a flamed needle or scalpel remove some of the spores from specimen or culture provided and stir them into the drop of water.
6. With a pipette run water, (e), about the inside of the petri dish and carefully drop the "lid," (f), into place over the slide. This water should be sufficient to seal the moist chamber. Additional water may be added between the lid and bottom if necessary after the cover is on.
7. Set away carefully in your locker or ice box, as may be required, for 24 to 48 hours.
8. To examine the culture, remove the lid and place the bottom with slide in place on stage of microscope. Examine with low power without cover glass to determine if germination has taken place.
9. To study under high power, remove slide, cover with cover glass slide and place on microscope stage. The addition of a little eosin at the edge of the cover glass will often help to bring out details of germtubes.
10. Such slides may be preserved for future study by placing a drop of mounting fluid at the edge of the cover glass and allowing it to slowly run under the cover as the water evaporates.

Illustration. Cross-section of petri dish moist chamber.

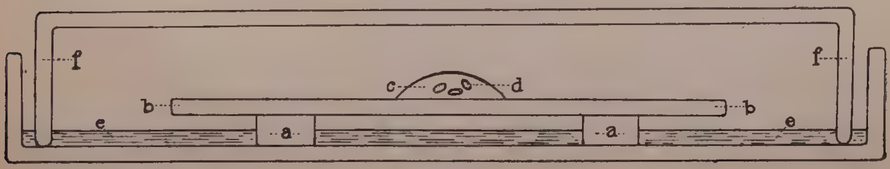

Fig. 1.

Directions for Germinating Sporangia of Phytophthora Infestans*

Phytophthora infestans (Mont.) DeBary, the cause of the Late blight of potatoes, belongs to that group of fungi known as the Phycomycetes. This group is characterized by having the asexual spores (swarmspores) borne in a sporangium. In the case of Phytophthora, as well as in *Plasmopara*, the sporangia most commonly germinate by means of swarmspores, although germination direct by a germtube is possible and is determined by either (1) age or a tendency innate within the sporangia themselves or (2) external influences, such as temperature, oxygen supply, moisture, light, or the concentration or nature of the medium in which they are placed.

According to Melhus (1915 : 57) and Murphy (1922 : 454) temperatures below 20° C have been found more favorable for *indirect germination* (swarmspore) in water, than are higher temperatures. The minimum lies between 2° and 3° C, the optimum between 12° and 13° C, and the maximum between 24° and 25° C. With good material the germination runs from 50 per cent. to nearly 100 per cent. An abundant supply of oxygen and the absence of competing organisms favor swarmspore production. *Direct germination* (by germtube) was found to be very scanty below 15° C, (with exceptions). Above 20° C direct germination became more abundant, increasing with the temperature. The minimum for this type of germination is probably between 10° and 13° C, the optimum about 24° C, and the maximum very near 30° C. Germination of this type seldom exceeds 50 per cent. and is generally less. Murphy holds that the tendency to produce germtubes at low temperatures or swarmspores at higher temperatures is innate in the conidia themselves and not governed by environmental conditions.

The time required for the germination of sporangia depends upon two factors:—(1) their viability and (2) external influences. The shortest period for indirect germination reported by Melhus was 45 minutes. Murphy (1922 : 455) states that if sporangia, when placed in water do not germinate within twenty-four hours, they seem practically incapable of producing swarmspores. Only hyaline sporangia will germinate; all dark colored or slightly plasmolyzed sporangia fail to grow.

Sporangia for germination may be secured from two sources:

(1) Petri dish cultures.

(2) Diseased plants showing the sporangiophores of the pathogene (from field or greenhouse).

In the case of petri dish cultures, suspensions may be made by removing the sporangia from the culture with a sterilized scalpel or needle and placing them in a watch glass or petri dish of sterile distilled water. After transferring a sufficient number of sporangia, place the suspension in the ice box for from 1½ to 3 hours at 10° to 14° C.

In case infected plants from the field or from the greenhouse are used, put the plants, pots and all into a moist chamber over night (or longer) until the sporangiophores are produced (avoid sprinkling water on plants, especially the under sides of the leaves).

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* The same methods with temperature variations to suit may be employed in germinating spores of other phycomycetous pathogenes.

Allow the foliage to dry. Remove the sporangia with a *clean, sterile* scalpel or needle. This may best be done by removing the infected leaf from the plant. Lay it down, moisten the point of the needle and collect the sporangia on the needle, without puncturing the leaf. Take sporangia from those regions only which have not become discolored or at least where the plant tissue is firm, at the margins of the spot.

Transfer the sporangia to *distilled water* in a clean dish (watch glass, petri dish, or flask). For indirect germination incubate for 1½ to 3 hours at 10° to 14° C in the ice box.

For best germination, the water should be neutral (P_H7, appearing grass green or a faint yellowish green).

Examine the spore suspension at the end of 1½ hours. If the sporangia are not germinating return them to the ice box and examine later.

Great care should be exercised throughout the work to prevent the introduction of toxic substances into the spore suspension, either from the suscept, dishes or microscopic slides or cover glasses.

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(2) *Plasmopara viticola.* (B and C) Berl. and De Toni.

Gregory, C. T. Spore germination and infection with Plasmopara viticola. Phytopath. **2** : 235–249, fig. 1–7. 1912.

DIRECTIONS FOR MAKING FREE-HAND SECTIONS

In the study of plant diseases, it will frequently be found necessary to make free-hand sections in the laboratory. This applies particularly to the study of histological symptoms and the morphology of the pathogene.

Each student should provide himself with the following apparatus from the stock room (in addition to the razor and pith furnished in the tool box):—three Syracuse watch glasses, one medicine dropper and one tumbler.

A sharp razor is issued in the tool box; use every *precaution to keep it in good condition.* A sharp razor is *absolutely essential for successful sectioning.* When dull, exchange for sharp one. Strop frequently.

Material for sectioning is of three kinds: (1) fresh, (2) preserved, (3) dried.

A. **Fresh material sectioned without the use of pith,** such as stems, roots, etc. In sectioning fresh material brace the forearms against the side of the body, hold the object firmly in the left hand, and cut with a long oblique stroke from left to right. When sectioning leaves fold several times. The edge of the razor and direction of the stroke should be toward the body, not away from it. The object and the edge of the razor blade should be kept *wet with water*. This may be accomplished by flooding the side of the blade, held uppermost, with the water (using a camel's hair brush) and gradually allowing it to flow onto the object.

Remove the thin sections from the razor blade to a watch glass by washing off with water by means of the medicine dropper, the camel's hair brush or with the thumb of the hand holding the razor. The watch glass with sections in water may then be examined under the low power of the compound microscope and those thin enough for study transferred to a slide. Use great care to avoid tearing or injury in removing sections to slide from the watch glass. A loop, section lifter, needle, fine forceps, brush or even the medicine dropper may be found convenient for this purpose.

B. **To prepare pith for sectioning.** Remove the hard, rough outer layer of the pith for a distance of an inch or two back from the end of the pith stick, with a sharp scalpel blade, (*not with the razor to be used in sectioning*), and place in a bottle of water to soak. Allow to soak from a few (5–10) minutes to several hours. Then make a narrow slit in the prepared end of the pith stick with the sharp scalpel. The pith is now ready to receive the object to be cut.

C. **To prepare object for sectioning in pith.** 1. *Fresh material:* Such as leaves, parts of stems or fruits or sporophores. Cut the material to be sectioned into small conveniently sized pieces and insert the piece or object into the proper position in the slit prepared in the pith stick and hold and section as directed in A above. In sectioning leaves several layers made by folding should be inserted in the pith slit.

2. *Preserved material:* When material is preserved in liquid follow directions as in C, 1, with the following exception. When the material is preserved in formalin wash the object in water to avoid the odor of the fumes.

3. *Dried material:* Arrange the three watch glasses in a row upon the desk. From the reagent bottles pour a small amount of 70 per cent. alcohol into watch glass number one; some potassium hydroxide (KOH) solution (7 per cent.) into watch glass number two and water into watch glass number three. From the dried specimen cut a small piece of the desired material for sectioning, place the object in the 70 per cent. alcohol for a few minutes to remove the air; then transfer to KOH for a few minutes ($\frac{1}{2}$–5) to swell, soften and clear it; then transfer the object to water and wash out the KOH. The edge of the razor blade will be injured if KOH is left in the object. Place the object in the prepared pith stick and section as directed in A. The object and the edge of the razor should be kept flooded with 70 per cent. alcohol or water to avoid tearing the section. The sections may be washed from the razor blade into the watch glass containing the 70 per cent. alcohol or water by means of the medicine dropper, the brush, or with the thumb; the pith sections removed with the forceps and the sections examined under the low power of the microscope to locate those thin enough for study. Mount the sections on slides, using forceps, needle or brush.

D. **Staining.** A simple method to differentiate fungus and suscept tissue is by the use of eosin. Mount the section on a glass slide in water and stain by placing a drop of eosin at one side of the cover glass, drawing the water off at the other side by means of a blotter or piece of cheese cloth. After staining a few minutes (1–5), depending upon the material, remount in water in the same manner that the eosin was introduced. This should stain the fungus a pink to red, whereas the tissues will retain very little of the stain. Iodine and other stains may be used in the same manner.

GLOSSARY*

Agent of inoculation.—The thing which acts as the carrier in the transfer of the inoculum from its source to the infection court, as for example:—wind, insects or running water.

Control.—The prevention of losses from a disease. Every control measure is based on one of four fundamental principles; exclusion, eradication, protection or immunization.

Dissemination.—The act or manner of scattering or spreading the inoculum of the pathogene within the immediate and more or less limited area about the source of inoculum.

Distribution.—(a) The act of transporting and establishing a pathogene beyond barriers into other regions. (b) The geographical occurrence of the disease (synonymous with *Range*).

Enphytotic.—The opposite of epiphytotic. An enphytotic disease is one regularly occurring in a locality or region and not liable to marked variations in destructiveness.

Epiphytology.—That phase in the study or discussion of a disease which deals with the relation of environmental factors to its occurrence, severity and character. These factors are chiefly climate, soil and culture. They influence the disease indirectly through their influence on the pathogene or the suscept or on both.

Epiphytotic.—The sudden and destructive appearance of a plant disease in a locality or region. An epiphytotic disease is one the past history of which shows it to have a tendency to appear suddenly and destructively, usually over large areas at rather long intervals. The term is analogous in meaning to epidemic but not synonymous with it.

Eradication.—The principle of controlling a plant disease by removing or destroying the pathogene already established within a given area or region. Disinfection of soil, cultivation, crop rotation and the like are eradicatory measures.

Etiology.—That phase in the study or discussion of a disease, which deals with the chief causal factor, its nature, classification and relations with the suscept.

Exclusion.—The principle of controlling a disease in plants by excluding the pathogenes from a given area or region. Seed disinfection, inspection and quarantine are the exclusionary measures usually employed.

History of a disease.—The logically arranged historical facts, relating to the disease itself, as distinguished from those relating more especially to the pathogene.

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* These definitions apply to the respective terms only as these terms are used in a phytopathological sense, and particularly as used in these outlines. The definitions do not always agree with those to be found in dictionaries, nor always with the variety of senses in which the terms are used even in phytopathological literature.

Host.—Any plant subject to attack by a parasite. The use of the terms host and parasite connote a food relation not a pathologic relation. The universal use of these two terms in the latter connotation is unfortunate and misleading. (See *Suscept.*)

Hyperplasia.—That type of pathological condition expressed by abnormal increase in the number of cells, that is, excessive cell division.

Hypertrophy.—That type of pathological condition expressed by abnormal increase in size of cells (dimensions or volume).

Hypoplasia.—That type of pathological condition expressed by subnormal cell multiplication. Dwarfing is an example.

Immunization.—The principle of preventing losses from a plant disease by the development of resistant or immune strains of the crop. This may be accomplished by selection and propagation of naturally resistant or immune individuals, by segregation and propagation of resistant or immune individuals obtained by crossing immune and susceptible forms or, artificially by feeding or injecting into the suscept substances which will make them resistant or immune. The last has, as yet, little or no practical value.

Incubation.—The activities and developments of the pathogene during the incubation stage.

Incubation period (of the disease).—See *Latent period.*

Incubation stage.—That stage of pathogenesis beginning with the arrival of the inoculum in the infection court and ending with initial infection.

Infect.—To initiate or produce disease.

Infection.—The act of producing or initiating a diseased condition in the tissues of the suscept. Infection is progressive; developing cell by cell and continuing as long as the suscept continues to react to the stimulus of the pathogene.

Infection court.—The place on or in the suscept where the incubation activities of the pathogene take place; the immediate neighborhood of a possible point of infection.

Infection period.—Commonly used to designate the period during which conditions (of suscept, pathogene, and environment) are especially favorable to inoculation, incubation, and initial infection, or to incubation and initial infection only. An unsatisfactory term for this concept.

Infection stage.—The stage of pathogenesis beginning with the initial reaction of the suscept to the activities of the pathogene and ending with the final reaction of the suscept thereto.

Inoculate.—To transfer the inoculum from its source to the infection court.

Inoculation.—The act of inoculating. It includes all the activities involved in the transfer of the inoculum from its source to the infection court.

Inoculation stage.—That stage of pathogenesis beginning with the dispersal of the inoculum and ending with its arrival in the infection court.

Inoculum.—That portion of the pathogene which may be transferred from its source to the infection court. It usually consists of spores, seeds, eggs, thalli (of bacteria), or even mycelial fragments or pieces of stems (Cuscuta).

Latent period.—The period of time during which the pathogene is becoming established in or on the suscept prior to the first visible evidences of disease. This is commonly spoken of as the incubation period. The latter is an unfortunate term for this concept.

Lesion.—A definite region in a plant or in one of its organs, characterized by a pathologic change in structure.

Life cycle.—The succession of phenomena exhibited during a period of continuous growth and development of the pathogene. (In a fungus, from spore germination to the normal death of the mycelium thus initiated.) Most pathogenes exhibit in their life cycles two rather distinct phases, pathogenesis and saprogenesis.

Life history.—The complete succession of phenomena characterizing a pathogene throughout the various cycles of its existence.

Metaplasia.—The type of pathological condition expressed by the overgrowth in cells, other than increase in size (hypertrophy) or numbers (hyperplasia). Abnormal starch accumulation, abnormal development of chlorophyl, unusual thickening of cell walls and abnormal nuclear division or growth are evidences of metaplasia.

Necrosis.—That type of pathological condition expressed by the rapid destruction of cell structures and a consequent prompt death of the protoplasts; rots, blights and cankers are examples of necrotic symptoms.

Pathogene.—Any organism capable of causing disease.

Pathogenesis.—That portion or phase of a life cycle during which the pathogene becomes and continues directly associated with the living suscept. Pathogenesis includes inoculation, incubation and infection.

Pathogenic disease.—A disease caused primarily by a living organism.

Pathogenicity.—The ability of an organism to produce disease.

Pathogenicity studies.—Experimental studies demonstrating the pathogenicity of the given organism.

Physiogenic disease.—A disease caused primarily by some non-living factor, as frost, water, heat, etc.

Primary cycle.—A life cycle involving a primary infection.

Primary infections.—Those infections first initiated by the pathogene after a period of rest or relative inactivity. In temperate regions, pathogenes usually initiate their primary infections in spring or early summer.

Protection.—The principle of controlling a plant disease by placing some protective barrier between the suscept and the generally present pathogene. Spraying, dusting and coating with substances inimical to the inoculum of the pathogene but harmless to the suscept are the usual protective measures employed.

Range.—The geographical regions, areas or countries in which the disease is known to occur.

Saprogenesis.—That phase of a life cycle during which the pathogene is not in direct association with the living suscept. Some pathogenes exhibit no activities during saprogenesis.

Secondary cycle.—A life cycle involving a secondary infection.

Secondary infections.—Those initiated by inoculum from the primary or other secondary infections without an interposed resting or dormant period, i. e., during the same growing season.

Signs.—Incidental or experimental evidences of disease as distinguished from pathological evidences. The usual signs of disease are the structures of the pathogene produced in or on the lesions.

Sources of inoculum. The place or object on or in which the inoculum is produced, as some organ of the suscept or some object of the environment.

Suscept.—Any plant liable to infection by a given pathogene. This term is here coined to take the place of the word host when considering the pathological relationships of the causal organism and the plant which it affects. (See *Host.*)

Symptomatology.—That phase of plant pathology which deals with the signs and symptoms of disease for purposes of diagnosis.

Symptoms.—Those expressions by the suscept of a pathologic condition by which a diseased plant is distinguished from a healthy one.

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